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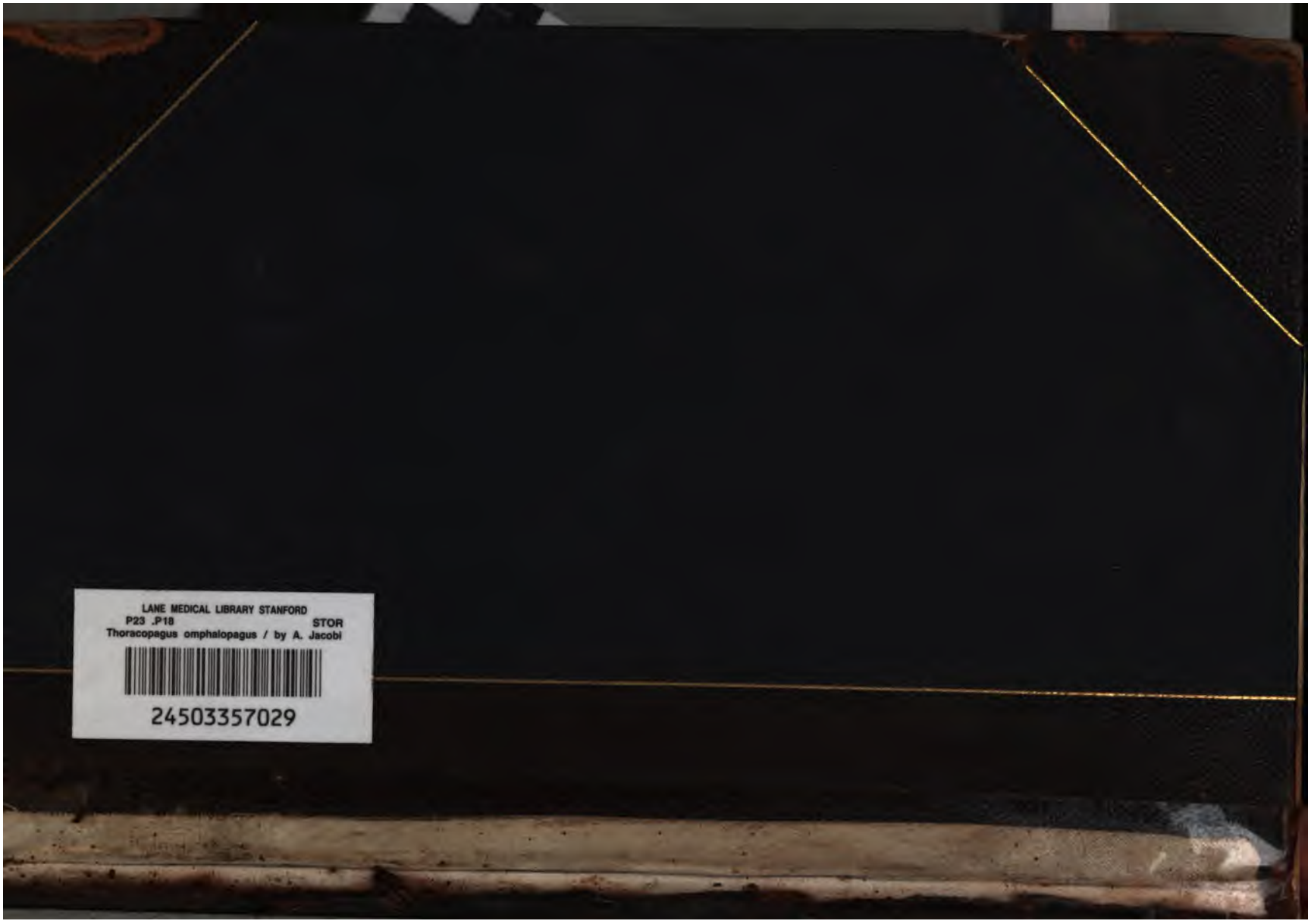
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BY

J. HENRY FRUITNIGHT, A.M., M.D.

FELLOW OF AMERICAN ACADEMY OF MEDICINE, FELLOW OF NEW YORK ACADEMY OF MEDICINE, SECRETARY OF PÆDIATRIC SECTION OF NEW YORK ACADEMY OF MEDICINE, ETC.

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SCARLATINOUS OTITIS.

BY

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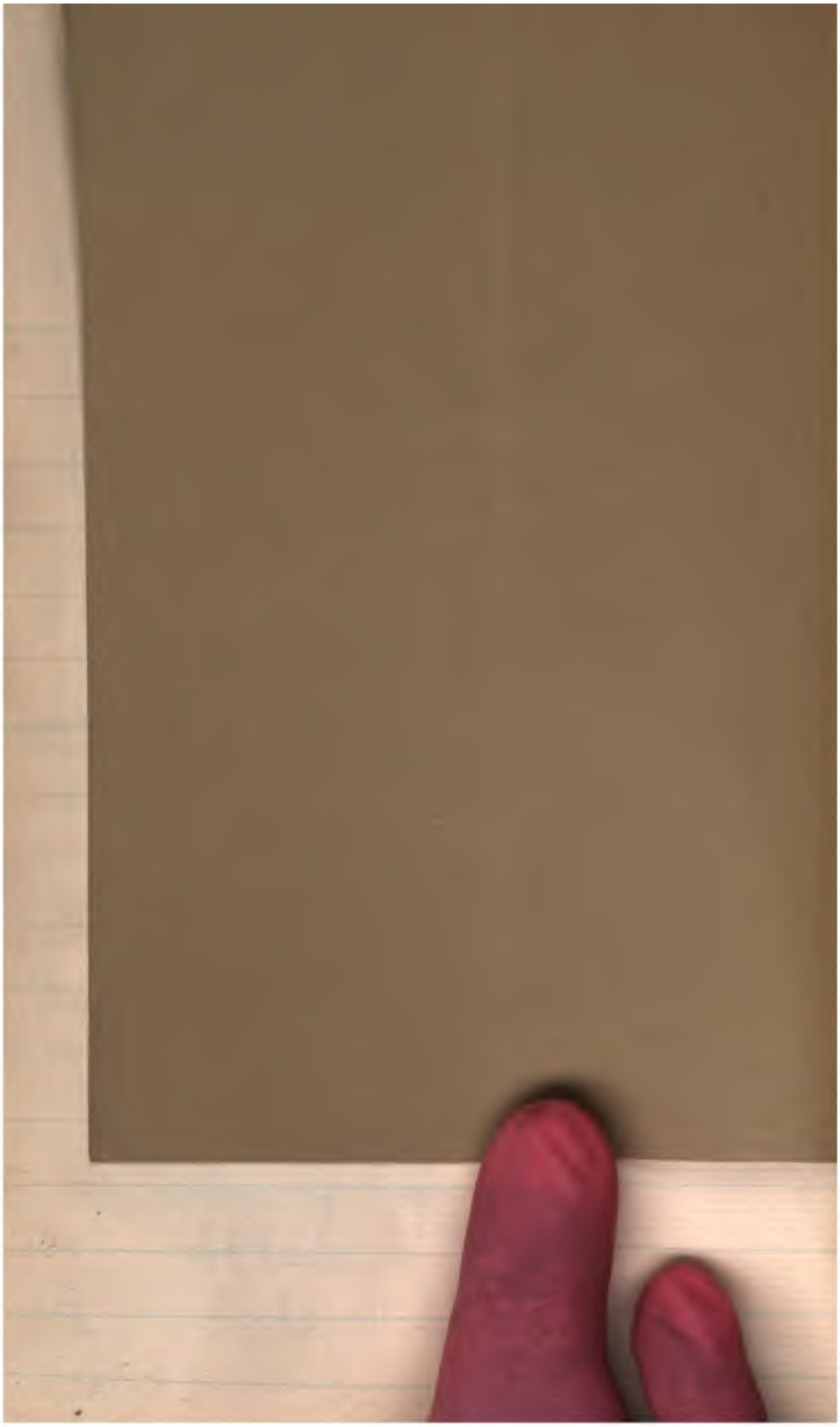

URINARY CONCRETIONS IN CHILDREN.[1]

By J. HENRY FRUITNIGHT, A.M., M.D.,

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ETIOLOGY.—Though urolithiasis may appear to be relatively an infrequent disease in childhood when compared with the whole number of children living, yet it is of special interest because the greater number by far of all the cases of this description is found among children. It is said that more than half of all the cases of calculus—and the vesical variety is the one especially meant—occur in childhood. Furthermore, when it occurs in children it is most frequent in those under five years of age. Nor are renal calculi so very infrequent in children, for the accumulation of uric acid in the kidney soon after birth may prevent the excretion of urine and may sometimes cause the formation of stones, which are occasionally found in very young infants. Thus Professor A. Jacobi has recorded that six times in forty post-mortem examinations of children under one year of age he has found renal calculi.

According to Roberts, of Manchester, England, of 1,827 persons who underwent lithotomy at various hospitals in England, 473 were under five years of age, and 528 were between five and fifteen years of age, hence more than fifty per cent. of these cases occurred before

[1] Read at the Stated Meeting of the Northwestern Medical and Surgical Society of New York, January 16, 1889.

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puberty. Dr. Geo. E. Post, of Beirût, Syria, has tabulated 176 cases of vesical calculus occurring in his practice, and 106 of these were in children under ten years of age.

As in adults, calculi are more frequent in the male than in the female sex. Barthez and Rilliet cite eight hospital cases, of whom six were boys and two girls. Neubauer found but five girls in one hundred cases of the disease.[2] Giraldes asserts that vesical calculus happens twenty-four times more frequently in boys than in girls.[3]

Though the fact is well established that the disease is more prevalent in some regions of the world than in others, as in the warmer zones for instance, yet no portion of the globe seems to be totally exempt from its presence. The negro race, however, is claimed to be remarkably free from calculous disease.

It has been observed that those children in whom calculi are found usually belong to the humbler classes, just the reverse of what obtains in adult life. According to the writings of Sir Henry Thompson half of the cases admitted to Guy's Hospital for operation for stone are children of the poor. He states, too, that it is almost never found among the children of the more favored classes. This has been explained by the fact that in consequence of the coarse and indigestible food of which they partake, and which is chiefly amylaceous in character, the children of the lower classes suffer from malnutrition, which is favorable to the deposition of urinary sediments. As more care and attention are paid to the diet and nutrition of the children of the better classes this cause is not operative in them. The absence of milk as an article of diet is supposed by some to tend to the formation of stone.

Scrofulous and tubercular children have also been con-

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[1] Transactions of Ninth International Medical Congress, vol. i., p. 610.

[2] Jahrbuch der Kinderheilkunde, 1872, v., Heft 4.

[3] Cyclopædia of Obstetrics and Gynecology, vol. x., p. 192. New York: Wm. Wood & Co. 1887.

sidered as predisposed to this affection, and in particular rachitic children are claimed to be remarkably prone to be affected. Of the eight cases of Barthez and Rilliet, already alluded to, four were afflicted with tuberculosis of the encephalon, and one with acute tuberculosis associated with pulmonary gangrene. Vesical calculus has also been announced as among the sequelæ of measles.[1]

Heredity and family idiosyncrasy also seem to exert an influence in the occurrence of this disease, though by no means constant. A writer in the *London Lancet* for July 10, 1872, on page 204, reports a curious instance of this: "Six children of one family all had stone; the father and mother both passed quantities of uric acid, the grandfather and grandmother, a great-uncle, six uncles, four aunts, and one cousin had either all had attacks of gravel or were cut for stone."

Habits of life and nutrition play quite a prominent part in the causation of lithiasis. Habit naturally plays a more active part in adult life than in children, for the latter have not yet been confirmed in their habits, and it is therefore virtually without influence in early life.

Poor nutrition, already alluded to, favors the occurrence of lateritious deposits because of the abnormal composition of the urine under such circumstances.

The activity of the skin also enters as a factor of some influence in the production of urolithiasis; for the more active the skin is the more concentrated will the urine be, therefore the liability to calculus is increased because this liability stands in direct ratio to the amount of solid ingredients, and especially to the amount of uric acid which the urine contains.

The same general intrinsic causes are equally active in the child as in the adult. The concretion may be deposited in some one of the urinary organs from the unchanged urine because of some inherent vice in its composition. For instance, there may be present an excessive proportion of precipitable ingredients in the urine. The

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[1] Coulson: Jahrbuch für Kinderheilkunde, No. 440, p. 34.

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urine may be too acid in reaction, thus diminishing its solvent power over uric acid and the urates. A deficiency of the chloride of sodium and the alkaline phosphates also reduces its power of holding uric acid in solution. Again, an ammoniacal decomposition of the urine in the lower urinary passages is favorable to the precipitation of the earthy phosphates and carbonate of lime. Acid fermentation of the urine with the mucous secretions of the urinary passages assuming the rôle of the ferment, is another cause of urinary concretions. Occasionally children may have a catarrhal condition existing in the pelvis of the kidney which will favor the formation of uric-acid gravel, and which may also lead to symptoms which correspond exactly with those of calculus pyelitis of later life. If this catarrh of the pelvis of the kidney should last a long time it might gradually spread through the ureter to the mucous membrane of the bladder, and as vesical catarrh notably favors the formation of stone, vesical calculi might be developed.

The presence of an abnormal substance of slight solubility in the urine, as cystine or xanthine, predisposes to the formation of calculi. The accidental presence of a body suitable to become a nucleus for the crystallization of urinary sediments, as a clot of blood, inspissated mucous epithelium, etc., or a foreign body introduced from without, as a needle, a piece of wire, a bone, etc., will also give rise to the formation of concretions. I do not, however, know of any case among children which originated in consequence of the introduction of a foreign body from without.

That certain diatheses—as the rheumatic, gouty, or dartrous—exist in children, as in adults, favorable to the occurrence of these urinary deposits is to my mind problematical.

Pathology.—These concretions are most generally formed in the kidneys, whence they often pass into the bladder, where they increase in size and weight, and occasionally into the urethra. They are very seldom indeed formed in the urethra, but if found there they have been

almost invariably arrested in their passage from the kidneys. They also may occasionally originate in the bladder. Urethral calculi occur especially in children. As has just been remarked, they are derived for the most part from the bladder or the kidneys, being calculous fragments which have been arrested at the narrowest part of the canal and lodging more especially at the bulbous portion or fossa navicularis. In some very rare instances the concretions may be formed in the urethra. Thus there may be a constriction in the canal behind which the urine may collect and deposit some of its salts—a nucleus is formed, usually of uric acid, and the evolution of a urethral calculus has begun. Again, the arrest of one or more gravelly particles may also give rise to the formation of urethral calculi. Dr. Fewsmith, in vol. xv. of *The American Journal of Obstetrics*, refers to a case of primary urethral calculus in a boy one month old which was apparently of intra-uterine growth.

Concretions may also form just within the prepuce, particularly if the latter be long and tight, and hence they are known as preputial calculi. These calculi may consist of inspissated smegma or of true urinary deposits. The latter variety may either have been formed here in a manner similar to what obtains when a urethral calculus develops *in situ*, or it may have escaped from the urethra into the preputial canal.

In newly born infants uric-acid infarctions are nearly always found in the kidneys. These are insignificant in extent, and they very rarely disturb the renal functions. The opinion which formerly prevailed, that these infarcts of uric acid were found exclusively in children who had breathed, has been demonstrated to be false. Indeed, they have been observed in the kidneys of still-born children, and even in those of the non-viable fœtus.

The presence of these infarctions in newly born infants is explained as being caused by an increased physiological tissue metamorphosis, which is induced after birth by the establishment of the processes of respiration, digestion, and calorification. These infarctions are rarest in those in-

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fants who die very soon after birth. They are more frequent in those children who have lived twenty-four hours, and most frequent in those who die between the second and fourteenth day after birth. Then until the end of the second month there is a steady diminution in the number of cases presenting specimens of these uric-acid infarctions, though Virchow has remarked their presence even in the period extending from the third to the fifth month of life. They are to be found, however, only in the minority of the kidneys of the newly born. In fact, these infarctions are met with in less than fifty per cent. of the newly born infants, which proves that the conditions assuring their production must be inconstant. Concretions have also been detected in the kidneys in fœtal life. Langenbeck has discovered a calculus in the bladder of a six-months fœtus, demonstrating that they may be formed in intra-uterine life.

The calculi met with in children are most commonly composed of uric acid or the urates. Those consisting of the urates, and especially of the urate of ammonia, are most frequently found in nurslings. Ebstein denies the assertion that oxalic-acid concretions are more frequent in children than in adults. He refers to the case of a girl, seven years of age, who passed two large calculi whose kernels were uric acid, though their shells and middle layers consisted of oxalate of lime. If the urine be very concentrated, very hard uric-acid concretions will be produced by the depositions of concentric layers of that substance, and which may vary in color.

Cystine concretions are very rare in children, although Hodann removed a large one of this variety from a boy, six and a half years of age, by lithotomy. Xanthine and fibrine concretions, so far as I am aware, have not been observed in children; nor, according to Heller, have true phosphatic stones or the earthy phosphates been found in children. Carbonate-of-lime calculi, which are so extremely rare even in adults, have never been seen in children. When the concretions are formed in the urethra they are, as a rule, phosphatic in composition.

SCARLATINOUS OTITIS.[1]

WERE any excuse necessary for the presentation of the paper which I have the honor of having been asked to read this evening, it could be found in the large number of cases of impairment of hearing and of total deafness resulting from scarlatina, and in the fact that the dangerous opinion crediting scarlatinous otitis with being able to take care of itself and needing no treatment is quite prevalent. Some remarks on this subject seem quite opportune, now that a great many cases of scarlatina are being reported.

Though ear affections as complications or sequelæ of scarlatina are known to be quite common, I have been able to find but one author who gives expression to this frequency in figures: Burckhardt-Merian[2] quotes Baader, who observed this frequency in two epidemics of the diseases; in one 33%, in the other 22% of all cases presented otitis as a complication or as a sequel to scarlatina.

Much more statistical evidence is at hand to show in what a large percentage of the cases which present themselves with ear affections, scarlatina has been responsible for the aural trouble. Thus Schmalz reports 2,500 cases of ear affections, of which 137 were due to scarlatina (5½%). Jearsley found 26 cases due to scarlatina among 544 examined (almost 5%). Burckhardt-Merian, among 1,950 cases of ear disease, found 85 due to scarlatina (4⅓%). About 5%, therefore, of all aural affections appear to be the result of scarlatina.

[1] Read before the Pediatric Section of the New York Academy of Medicine, March 14th, 1889.

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When we come to examine the nature and extent of these ear affections due to scarlatina, we find that not only are they numerous, but the percentage of serious cases in which marked impairment of hearing, or even total deafness, is present is a large one. Burckhardt-Merian found entire destruction of the membrana tympani in 34% of cases of scarlatinal otitis; and as an additional illustration of the serious effects of otitis after scarlatina, when left to itself, the following brief resumé may be of interest: Bezold [1] reports 185 cases in which these effects after scarlatina were carefully observed:

Of these 185 cases, in 30 the entire membrana tympani was destroyed, with loss of one or more bones; in 59, the perforation comprised two-thirds or more of the membrana tympani; in 13, there were smaller perforations; 44 cases were complicated with granulations and polypi.

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But the most impressive idea of the alarming effects of scarlatinous otitis upon hearing may be gathered from the following table, which has been taken from the monograph of Burckhardt-Merian already alluded to, and to which additions have been made. In its modified form, this table represents reports from deaf-mute institutions of many different countries:

Reported by.	Total Number of Cases of Deaf-mutes.	Number of Cases Resulting from Scarlatina.	Percentage.
Toynbee	98	36	36.73
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Thus 10% of deaf-mutes owe their affliction to the effects of scarlatina. The study of these figures should certainly result in diminishing the tendency on the part of the general practitioner to do nothing in the way of treatment in cases of scarlatinal otitis. There was a time, many years ago, when the discharge of scarlatinal otitis was looked upon with feelings of satisfaction and regarded as a favorable means of ridding the system of the scarlatinal virus; but the days of such erroneous pathological ideas are past; the reason for inactivity in the treatment of scarlatinal otitis by the general practitioner probably is that he regards the achievements of otology as doubtful and unsatisfactory. Though this may be the case in some affections of the ear, in the group of cases comprised under the title of this paper, aural therapeutics are capable of producing the most happy results; and there is no doubt that the active treatment of these cases by the general practitioner, during the course of scarlatina, would do much to diminish the large percentage who owe a serious defect in hearing, or total deafness, to the effects of this disease. We should also remember that no patient is safe who suffers from chronic purulent otitis media; for we can never be certain when the disease will extend to neighboring parts, and when a serious or fatal result, such as meningitis or cerebral abscess, will ensue.

The symptoms which we get in scarlatinal otitis do not differ from those which we find in otitis occurring under other circumstances; they are apt to be severe and well marked. Usually the otitis does not occur until after the scarlatina has lasted several days, very often during the commencement of desquamation. We then often find the complication ushered in by chills; in very young children there may be convulsions; there is rapid elevation of temperature, often rising to 104° or 105°, with a corresponding increase in the frequency of the pulse and of breathing. There is great pain, at first paroxysmal and confined to the ear, but soon becoming of a steady neuralgic character and radiating in various directions from the ear, following branches of the fifth nerve; the pain is regularly worse at night. There is tenderness which may be confined to the region around the tragus, but often extends and involves the mastoid region, even in cases in which mastoiditis does not occur. Deafness is nearly always well marked and develops rapidly; both ears are affected in the great majority of cases,

child at once to the hospital, where the diagnosis was confirmed, and on the following day the patient was subjected to median lithotomy, which resulted successfully in the cure of the disease.

If the stone lie loosely in the bladder, the child will, in order to escape its suffering and to pass its urine more easily, lie on the back or on the side, as the force of gravity will cause the stone to fall away from the neck of the bladder when such a position has been assumed. Sometimes, if the stone be free and small enough, and provided it assumes a proper position as regards the relative direction of its longest axis and the long axis of the urethra, the calculus may be expelled spontaneously. Such a case came under my notice last year.

E. S——, aged seven years, complained that at times, when he was urinating the flow would suddenly cease, which was accompanied by pain. Straining did not cause a return of the flow. After he had run about for a time the urine would again be voided. One day when he had retained his urine longer than usual, as he expressed it, "a longish white worm" passed from him. This body was undoubtedly a calculus, which was the diagnosis that had been made.

When the concretion is arrested in the urethra, which happens quite often in boys, the symptoms are mostly mechanical in their nature. Complete retention of urine always occurs. Œdematous swelling of the genitals then ensues. Frequently the calculi can be felt in the urethra by manipulation. Two cases of this description came under my observation within a short period of each other, whose histories I will briefly narrate.

Case I.—G. B——, at the age of two and one-half years, had had an obstruction to the flow of urine. He was examined by Professor Jacobi, who found a stone in the bladder which he removed by lithotomy. When the boy was nearly five years old I was called to see him because he could not pass his water. In examining him I detected under my fingers a hard body in the course of the urethra, about one-half inch behind the corona glandis, and which

caused the boy pain when it was squeezed. He was placed under ether, and by manipulation I was enabled to force it forward to a point within reach of the grasp of a long pair of dressing-forceps, with which the foreign body was extracted. It proved to be a urinary concretion.

CASE II.—G. K——, aged three years, was passing water when suddenly the stream stopped and a whitish body partially protruded from the urethra. His parents were much alarmed and sent for me. The case was at once recognized as a urinary calculus impacted in the urethra. It was easily removed from the canal with an ordinary pair of dressing-forceps. Both children were then treated for urolithiasis for an extended period of time, and no more gravel or stones were developed.

If the stone be impacted in the deeper portions of the urethra very serious results often follow. For if the retention of urine be prolonged and relief be not quickly afforded, the irritation may eventuate in abscess, sinus, and urinary extravasation in the perineum, and finally incontinence of urine will succeed the irritability of the bladder.

Diagnosis.—The diagnosis of renal colic in infants is manifestly difficult. It is very likely that often when intestinal colic has been diagnosticated the child is really suffering from nephritic colic. Hence it is proper when a child is very restless and its actions denote pain to examine its napkins for the reddish-brown stain characteristic of urolithiasis. If dysuria be present the napkins should also be examined for the lithic sediment. If small concretions should be found in the diaper or in the urine of older children, it is advisable to explore the urethra and bladder of the anæsthetized patient. Every case of chronic dysuria, whether it be associated with vesical catarrh or not, should be thus examined in order to determine the presence or absence of calculi, as the exciting cause of this continued difficult and painful micturition. The predisposition to lithæmia is only detected after gravel or concretions have actually been found.

When the stream of urine is abruptly checked we may

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reasonably suspect the presence of a stone either in the bladder or in the deep urethra. But if we are in doubt the bladder should be examined in the usual manner with the sound in order to clinch the diagnosis.

Prolapsus ani in older children is a symptom which should not be undervalued for its indication of vesical calculus as a possible exciting cause. Priapism, too, in small children, should draw our attention to the possible presence of stone in the bladder. The presence of blood in the urine of children should certainly lead us to suspect the presence of calculous disease.

When called to a child suffering from retention of urine always suspect a concretion impacted in the urethra. For in boys the impaction of calculi in the urethra is almost the sole cause of urinary retention. In order to verify the diagnosis a physical examination of the perineum, and per rectum, should be made. Furthermore, in order to test the permeability of the urethral canal, it should be explored with a probe, and thus the diagnosis will be conclusively settled. Finally, error in diagnosis from lack of symptoms is not likely to occur, because in children these are generally more obvious than in the adult.

Prognosis.—The prognosis of this affection is much more favorable in childhood than in adult life. Simple gravel is nearly always cured. Still the uric-acid infarcts of infancy may occasionally be the first cause of a secondary nephritis, this disease being the result of actual irritation or real injury inflicted by the gravelly particles in the urine. The attacks of renal colic always end in recovery. Nor when concretions of appreciable size have formed are they *per se* necessarily fatal. Their direct results may, however, tend to unfavorable complications which will modify the prognosis in special cases.

The disease is more fatal in the male than in the female sex. Dr. William Roberts states that in the nine years from 1857 to 1866, in England and Wales, only 116 deaths among children occurred from calculous disease, which, it must be admitted, is a very small ratio. The

rate of mortality of this disease among children is so low because they are usually seen early, and because proper measures of treatment can thereupon be instituted with more hope of success than later in life. Moreover, when operative procedures are resorted to, the prognosis is nearly uniformly favorable, for they are usually pre-eminently successful. Indeed, lithotomy in boys under the age of puberty is, perhaps, the most strikingly successful of all the great operations in surgery.

In regard to urethral calculi, it can be said that, if they are not located in the deeper portions, the prognosis is very favorable. If, however, they be seated near the bulbous portion, then the complications which may possibly attend them render the prognosis more grave, though not necessarily fatal.

Therapeusis.—The treatment of lithæmia in children is, as in adults, dietetic, medicinal, and surgical. In order to diminish the density of the urine, which, it will be remembered, encourages the lateritious deposits, and also to lessen the irritation of the kidneys, considerable quantities of water should be allowed. This recommendation applies as well to young infants as to older children. Parents generally do not seem to realize that the young child can become thirsty, and will require something to slake its thirst as well as older people, for we seldom meet with parents who give their infants water to drink. In older children other aqueous beverages and diluents may be permitted. Also, when from the previous voidance of concretions or gravel we are led to suspect a tendency to the formation of such deposits, we should, as a prophylactic measure, order copious drinking of fluids, the variety of which, whether alkaline or otherwise, must be determined by the chemical composition of the foregoing deposits.

The diet of older children should consist mainly of milk, the white meats, eggs, and farinaceous articles of food. Still the fear of azotized substances should not be carried too far in the regulation of the diet. Our object, in other words, should be to limit but not to restrict the

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ingestion of nitrogenous foods. Naturally the foods permitted must be easy of digestion.

Warm baths are to be recommended, because of the increased nutritive changes promoted by them. The bowels should be kept free. Whatever induces to an improvement in the general hygienic environment of the patient, including good air, food, clothing, etc., should be strongly insisted upon.

When acute paroxysms of nephritic colic attack infants and children they must be cut short by the exhibition, in sufficient doses, of the weaker preparations of opium, such as the camphorated tincture or papaver syrup.

Medicinal treatment should be directed toward the prevention of the formation of concretions when a tendency thereto is apparent, and toward the solution and consequent facility of expulsion of concretions which have already been formed. Hence it has been designated as the solvent treatment. The solvent treatment consists in the administration of alkalies or acids, according to the solubility of the deposits in alkalies or acids. In carrying out the solvent treatment, when the urine abounds in uric acid and its compounds, which is the usual condition in children, we must bring it up to a certain degree of alkalinity by the continuous administration of alkaline remedies, for these medicines are somewhat slow in their action. Among the alkaline remedies the acetate of potash and the citrate of potash are well thought of. The carbonate and citrate of lithia may also be given, likewise the carbonate of potash. All these medicines should be largely diluted with water in their administration, and the doses apportioned to the age of the patient. The phosphate of soda and ammonia, in doses of from gr. ij. ad gr. v. ter in die, according to age, has also been prescribed with benefit. The phosphate of soda—which also acts well on the bowels of children—benzoic acid, and the benzoate of soda may be given in the same doses. Nor should the liquor potassæ be forgotten. The alkaline mineral waters, such as the natural Vichy, Bethesda, clysmic,[1] Buffalo lithia, and others may also be recom-

mended. The oxalate-of-lime concretions also demand an alkaline treatment. So long as the urine when voided continues sweet, the alkalies may be given, but should it become ammoniacal, then they must be discontinued lest a deposit of the earthy phosphates occur. The milder diaphoretics and diuretics can also be advised.

In phosphatic deposits the acid treatment is the proper one; but as these deposits are not met with in children its consideration will be dismissed with these few words.

In children these solvent remedies should always be administered per orem. Though vesical injections may be feasible in children, it seems to me that, inasmuch as we can obtain all desired therapeutic effects by the internal administration of the medicines, such injections should be discountenanced. Besides the direct action of these solvents upon the delicate mucous membrane of the child's bladder involves a certain amount of danger to it.

We may conclude, therefore, that the solvent treatment is applicable in cases of simple gravel, and in the early period of the formation of concretions, but that it ceases to be so when the urine becomes ammoniacal, and secondary deposits accumulate on the surface of the stone.

But when the stone, and the uric-acid variety in particular, is of considerable size, the solvent treatment by medicines will avail nothing, and recourse must then be had to the art of surgery. As a detailed description of the various resources which the science of surgery has developed for the cure and relief of these maladies is not embraced in the scope of this article, I will content myself with merely calling attention to certain salient conclusions, which are the outcome of the experience and observation of many distinguished and skilful surgeons.

Neither the operation of nephrotomy, for the removal of a calculus impacted in the kidney, nor the operation for the removal of one impacted in the ureter has ever, so far as I can learn, been done in the person of a child. The question of its feasibility I cannot discuss, but must submit it to the judgment of the surgeon.

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In the past the operation of median lithotomy for vesical calculus was preferred for male children, as it gave the best results. For female children, dilatation of the urethra, with subsequent lithotrity, was the chosen mode of operation. Recently published reports, however, seem to show that the operation of litholapaxy is growing in favor as a promising operation for both male and female children, and it may in time supplant the two first-named operations for the majority of cases of vesical calculus.

When a concretion has become impacted in the urethra a gush of urine will sometimes force it out. If it remain fixed, the urethra should be dilated, and the offending body should then be extracted with proper forceps. If, however, the stone be of large size and deeply seated, an incision in the urethra may be necessary in order to facilitate its extraction. Care should be taken that it be not pushed back into the bladder. When the concretion has ulcerated through the urethra, incise the resulting abscess, remove the stone, and then treat the wound on general antiseptic surgical principles. The treatment of preputial calculi is so obvious that it does not need elucidation. Yet it may be proper to suggest the advisability of circumcision in certain cases of redundant prepuce, in order to prevent their formation.

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mastoid process and to the labyrinth also occurs, though fortunately in only a minority of cases. It is probable that in all cases of scarlatina congestion of the mucous membrane of the tympanum takes place; this may give rise to no subjective symptoms, and is probably devoid of consequences. In the milder cases, catarrhal inflammation exists, and the symptoms of this may be slight, and the complication may subside with little or no treatment after the angina, of which this is but an extension, disappears.

Though some cases of catarrhal otitis media give rise to marked symptoms, it is generally the purulent and diphtheritic varieties of the inflammation which furnish the cases of pronounced scarlatinous otitis. In these cases there is now no longer any doubt that the complication arises through a direct extension of the throat lesion through the Eustachian tubes into the middle ear; the theory of metastasis which was formerly held is now no longer tenable; direct extension of the pharyngitis accompanying scarlatina has been proven to be the mode of occurrence by numerous post-mortem sections.

It is now generally believed that the severer forms of scarlatinous otitis are due to a diphtheritic otitis media, and this even in cases in which diphtheritic patches cannot be observed in the throat; it is certain that those cases of scarlatina which are complicated by diphtheria are most likely to be affected with severe forms of otitis media—varieties which are remarkable for their malignant character, so to speak. Thus it is noticed in the severe forms of scarlatinous otitis, that a very rapid and extensive destruction of tissue takes place, more rapidly and more extensively than can be explained by maceration of the membrana tympani by the products of inflammation, and by pressure of the latter and the incarcerated secretions; for these conditions of maceration and pressure also exist in ordinary cases of otitis media purulenta, and yet such a rapid destruction of membrana tympani and loss of ossicles is not seen. Furthermore, even after spontaneous perforation, or after paracentesis, this peculiarly active destructive process continues; often in several weeks entire absence of membrana tympani and the escape of one or more ossicles are noted. Burckhardt-Merian found entire loss of membrana tympani in thirty-four per cent of cases of scarlatinous otitis which had not been specially treated. This same necrotic tendency is seen in cases of primary diphtheria

SCARLATINOUS OTITIS.

BY

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SCARLATINOUS OTITIS.[1]

WERE any excuse necessary for the presentation of the paper which I have the honor of having been asked to read this evening, it could be found in the large number of cases of impairment of hearing and of total deafness resulting from scarlatina, and in the fact that the dangerous opinion crediting scarlatinous otitis with being able to take care of itself and needing no treatment is quite prevalent. Some remarks on this subject seem quite opportune, now that a great many cases of scarlatina are being reported.

Though ear affections as complications or sequelæ of scarlatina are known to be quite common, I have been able to find but one author who gives expression to this frequency in figures: Burckhardt-Merian[2] quotes Baader, who observed this frequency in two epidemics of the diseases; in one 33%, in the other 22% of all cases presented otitis as a complication or as a sequel to scarlatina.

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though in a few the affection is unilateral. There is great restlessness; then the patient becomes quite stupid, and often, if there is no interference giving relief, symptoms indicating meningeal irritation, and even those of blood-poisoning, show themselves. There is usually swelling of the cervical, auricular, and submaxillary glands, and this swelling may precede the invasion of the otitis, or may have been present as the result of the scarlatina.

After lasting two or three days, these symptoms are usually relieved, even when no paracentesis is done, by spontaneous rupture of the membrana tympani; and following this there is a gradual diminution in the intensity of the symptoms, and they may cease entirely; or with the closure of the opening in the membrana tympani they may again become severe.

The diagnosis, though usually quite easy and apparent, is sometimes less evident. This may be the case in very young children who do not complain of the pain, and who give us no local symptoms referable to the ear, except perhaps occasionally to put the hand to this organ. In such patients, when, whilst the symptoms of the scarlatina are on the decline, there is an increase in temperature, restlessness, and other symptoms of irritation for which we can find no cause, the ears should always be examined; such young children often press the ear affected into the pillow, and in this way give us a guide to the part causing the exacerbation of symptoms. It is a singular fact that pain is not always complained of, even in children old enough to express their ailings, and in this way the aural complications may be overlooked until meningitic or pyemic symptoms have arisen. I have myself observed such a case, in which, though on the alert for ear complications, a purulent otitis occurring during the desquamative stage of an attack of scarlatina in a child was overlooked until compelled to find a cause for rapidly developing stupor, chills followed by fever and profuse perspiration, and other symptoms indicating a probable absorption of septic material by the blood; paracentesis of both membranæ tympani in this case was followed by immediate improvement.

The title "Scarlatinous Otitis" includes all the ear affections which may occur as complications or as sequelæ of scarlatina; in the great majority of cases they are examples of otitis media, catarrhal, purulent, and diphtheritic; but extension to the

mastoid process and to the labyrinth also occurs, though fortunately in only a minority of cases. It is probable that in all cases of scarlatina congestion of the mucous membrane of the tympanum takes place; this may give rise to no subjective symptoms, and is probably devoid of consequences. In the milder cases, catarrhal inflammation exists, and the symptoms of this may be slight, and the complication may subside with little or no treatment after the angina, of which this is but an extension, disappears.

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of the middle ear—the existence of this can scarcely be doubted now. On the other hand, it is somewhat peculiar that though aural complications of diphtheria are not infrequent, they are generally of the catarrhal and purulent types, and are rarely diphtheritic.

It is easy to account for the occurrence of diphtheritic otitis in cases in which scarlatina is complicated by pharyngeal diphtheria; for, besides direct extension, each act of swallowing causes some of the infected air of the naso-pharynx to be forced into the middle ear; in these diphtheritic cases, after perforation, shreds of false membrane will be found adhering to the walls of the tympanum; these are difficult to tear off, and are accompanied by a little secretion of a sero-purulent nature and often quite offensive.

Though such serious consequences result from scarlatinous otitis, the prognosis is certainly largely influenced by treatment. This is one of the redeeming features of otology. Without treatment the effects of the severer forms of scarlatinous otitis are very disastrous to the function of hearing; with treatment, it has been shown the results are very favorable. The very mild cases in which the inflammation is of a catarrhal type often do well, and recover perfectly without any treatment. In cases in which the internal ear is affected, the consequent impairment of hearing is thought to be produced by the deposit and pressure of inflammatory products upon the ends of the auditory nerve; in these cases, numerous examples are reported in which the deafness has been unexpectedly recovered from in the course of two or three months, and sometimes even without any treatment whatever; in such instances it seems plausible to suppose this improvement to be due to absorption of deposited inflammatory products.

In regard to treatment, there are simple and effective means which can be used by the general practitioner, which will suffice to cure the complication in mild cases, reduce the severity and diminish the disastrous effects upon hearing in the more serious forms. At first, all forms may be treated alike. With the occurrence of pain, tenderness, deafness, and other symptoms indicating an extension of inflammation to the middle ear, a long, narrow ice-bag should be applied so as to cover the region immediately behind the auricle, and curving around the lower end of this organ upon the temporo-maxillary

region; if a layer of flannel be interposed between the auricle and ice-bag, these applications are not, as a rule, disagreeable, and are very often effective. Tincture of iodine often acts well, painted over the skin immediately behind the auricle.

In some cases, these applications of cold are not borne well; then we should use hot applications, covering the entire auricle with cloths wrung out of very hot water; over these we place a large wad of cotton covering the entire side of the head and a layer of oil silk on the outside; in this way heat is most effectively applied.

Whether hot or cold applications are made use of, we must endeavor to control the pain, which in many cases is quite intense and is apt to be pronounced at night. The best manner of accomplishing this is to instil hot salt water (℥ i. to Oi.) into the ear, allowing it to enter the external auditory canal from a fountain syringe held not more than a foot above the level of the ear; or it may be poured in by a teaspoon or a medicine dropper; a little tincture of opium or cocaine may be added to the salt water, but these do not materially increase the anodyne properties of these applications in which warmth is the chief agent in relief.

Large doses of antipyrin are also useful in controlling the pain, and if these do not answer, an opiate should be given; for it is essential that the patient be relieved from pain and restlessness. Rest is an exceedingly important factor in all these cases, and must be insisted upon, if this has not already been done in the treatment of the scarlatina. It must be absolute rest in bed.

Another simple and valuable method of giving relief in these cases is local bloodletting. Two leeches may be applied just behind the ear, if there be any tenderness over the mastoid region, or at the anterior limits of the tragus, care being taken to prevent entrance of the leeches into the auditory canal by filling this with cotton. The artificial leech of Heurteloup may also be used over the region in front of the tragus, its application behind the auricle being difficult on account of the unevenness of surface existing in this situation.

In very mild cases, where there is but congestion or a mild form of catarrhal inflammation, these simple means will suffice, and be followed by a disappearance of all symptoms referable to the ear. But if, in more severe cases, the symptoms do not

disappear, examination of the membrana tympani will probably show intense congestion or a bulging of this membrane, and paracentesis is then absolutely indicated. It is not desirable to wait until a large collection of secretion in the middle ear gives us a classical picture of a bulging drum membrane projecting into the auditory canal, and presenting a marked yellowish reflex indicative of the existence of pus within; such classical pictures are not common under the circumstances, for the pain, tenderness, swelling of the canal and the difficulty of making a thorough examination often compel us to be satisfied with but a hasty glance through the speculum. Paracentesis is indicated in all these cases, even where there is no physical evidence of anything but congestion of the drum membrane; the results of this operation are almost always immediate and gratifying, whether pus has been evacuated or whether the incision has merely allowed the escape of sero-mucus, or of only a small amount of blood.

In such cases the early performance of paracentesis cannot be urged too strongly; some mild cases may get well without it, but even in these cases the incision can never be called a mistake; for did it accomplish nothing more than the immediate relief of pain, it would still be entitled to be regarded as a valuable resource. If the auditory canal be kept clean, this small operation never does any harm; the wound in the membrane has a pronounced tendency to heal rapidly, and this often before such closure is desirable.

After paracentesis, if nothing but blood or serum or sero-mucus issue from the opening, the case may be left to itself, a little cotton being kept in the auditory canal so as to absorb the discharge. The cotton should be loosely applied. The patient should lie with the affected ear upon the pillow, if the trouble be unilateral. Even if the paracentesis has resulted in evacuating pus, the case very frequently needs no further treatment; the cotton, loosely applied in the canal, should be changed frequently; after several days the discharge, gradually lessening, may cease entirely; the opening in the drum-membrane heals perfectly, and the patient hears as well as ever.

But supposing, in a case in which paracentesis has been performed and pus has been evacuated, there are evidences that the discharge does not escape freely, as shown by attacks of pain and by a commencing offensive odor of the material from

the middle ear; then it will be necessary to empty the middle ear completely once a day by pressure from behind, and to cleanse the auditory canal several times a day. The middle ear is emptied by inflation with Politzer's bag or with the Eustachian catheter; the catheter is preferable, because there is no danger of forcing offensive material from the nose and naso-pharynx into the Eustachian tube and thence into the middle ear; but it can rarely be used in children under four years, and even in older children its successful application seems to depend upon the skill of the practitioner—not infrequently the child will not permit its use even in the hands of the most experienced aurist. On this account the Politzer bag must be resorted to in a great many cases, the child being asked to swallow water, or in very young children being made to scream as a means of causing closure of the palate, and thus compelling the air to enter the Eustachian tube. Previous to Politzerization, it is well to wash out the nose and naso-pharynx; this can readily be done without any risk of exciting additional aural trouble by using an ordinary straight-pointed medicine dropper; the latter is filled with warm salt solution (℥ i. to Oi. water) and introduced into each nostril about half an inch in a horizontal direction; the bulb being compressed, the fluid passes through the nasal cavity and naso-pharynx, and escapes from the other nostril or from the mouth; this is an easy, safe, and effective manner of cleansing the nose and naso-pharynx, and I have never seen any injurious effects from its employment.

In those cases in which there is a tendency for the discharge to become offensive, or in which it is very profuse, it is necessary to keep the auditory canal clean. The very best solution for this purpose is an aqueous solution of salt—a teaspoonful of table salt to a pint of water; this should always be used warm. It is difficult to see why there should be any prejudice against washing out the canal in these cases, except that it may owe its origin to the fact that pure water is irritating if there be an opening in the drum-membrane, just as pure water is irritating to any mucous surface. But salt water of the indicated strength (¾ of 1%) is devoid of irritation, is cleansing, bland, and soothing. It is, of course, no disinfectant, but neither are boric acid in powder or boric acid or borax in solution; true disinfectant solutions are too irritating in this stage. The salt solution may be used either by fountain syringe (not

held too high, so that the force be not too great,) or the ordinary syringe used gently, undue pressure being injurious, and care being taken not to injure drum or canal by the nozzle of the syringe.

Often this treatment will be all that will be required; the opening in the drum-membrane, whether spontaneous or made by paracentesis, will heal after the secretion of pus has ceased. Packing the auditory canal with powdered boric acid in such a manner as to prevent the escape of pus is not only an irrational, but a dangerous proceeding, and may cause extension of the inflammation to deeper parts of the auditory apparatus; if the pulverized boric acid be blown in in quantities insufficient to cause a barrier to the escape of secretions, and the canal be washed out each time before the insufflation is repeated, no such objection can be urged against the boric acid treatment. The fact that after paracentisis these simple cases do well when kept clean, whether treated by insufflations of boric acid or where nothing but salt water is used, may explain why good results are reported after use of a great many different bland remedies; and these simple cases certainly do as well without as with boric acid insufflations.

Should a repetition of symptoms and an examination of the drum-membrane show that the edges of the incision have united, it is necessary to reopen the wound, or to repeat the operation of incising.

In performing paracentesis of the drum-membrane, it is customary to use a narrow knife resembling a lance-shape-pointed needle, and this is supported upon an obtuse-angled shaft; the speculum being introduced and a good light thrown in from the forehead mirror, an incision is made in the postero-inferior quadrant, or it may be made in any part of the posterior half of the membrane; the postero-inferior quadrant is usually selected because the tympanum is deeper here, and the opening is made in a part of the drum-membrane favorable for the escape of secretion. Any narrow knife, such as the Graefe cataract knife or a very narrow bistoury, will answer for this operation. The mistake usually made by those who do not perform the operation frequently is to make the opening too small; it can scarcely be made too large, and should be free and extensive, running from behind downwards and forwards. The operation is painful, but is only of momentary duration;

the most efficient way of reducing the amount of pain is to have the instrument employed very sharp, and to do the operation rapidly. Cocaine has some effect, but certainly not much in anesthetizing the part; and this does not seem surprising, when we remember that we are really dealing with a cutaneous surface.

It is not until all acute symptoms have subsided that it is proper to use astringents for the cure of any remaining otorrhea.

Where there are marked swelling and tenderness over the mastoid region—and these are not relieved by a paracentesis of the drum-membrane—it is proper to make Wilde's incision—an incision one or two inches long, extending in a curved direction corresponding to the posterior attached margin of the auricle, and from two-fifths to three-fifths of an inch behind this down to the bone.

My experience in the treatment of those severe and rapidly destructive cases of scarlatinous otitis in which the inflammation is frequently of a diphtheritic type has been rather limited, and therefore I prefer to give a brief resumé of the treatment recommended by Burckhardt-Merian, who is considered quite an authority in these cases. He advises the cauterization of the diphtheritic patches through the opening in the membrana tympani with a ten-per-cent solution of salicylic acid in alcohol; this he applies upon cotton at the end of a delicate cotton-holder, and such applications are made first twice a day, and then once daily; if patches are seen in the naso-pharynx these are also cauterized. The applications are painful, and this, as well as the tendency to vomit when the applications are made, is controlled by allowing the patient to suck small pieces of ice. The polypus snare and the aural curette are used to remove as much as possible of the diphtheritic membrane from the tympanum. The auditory canal is syringed several times a day with a solution of salicylic acid made in the proportion of one or two teaspoonfuls of a ten-per-cent alcoholic solution to 100 grams of water. A solution twice this strength is used for gargling. The nose is cleansed by the douche, first with salt water (three-fourths of one per cent), and gradually salicylic acid is added to this salt water in the proportion of two or three tablespoonfuls of this ten-per-cent alcoholic solution to one litre of salt solution. Sometimes he employs salicylic acid insufflations in the place of

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applications of the ten-per-cent solution. Other authorities have recommended filling the auditory canal with lime water several times a day, with a view to dissolving the diphtheritic membrane.

Where the nervous apparatus of the auditory organ has become involved as a result of scarlatina, our means of limiting this result are meagre, if we have any at all. Pilocarpine has been employed rather extensively for this purpose, daily hypodermic injections of one-twelfth to one-sixth grain being used for four or six weeks. The reports of the efficacy of this treatment are conflicting. Politzer, who first recommended the drug, is inclined to abandon it now; Rosengarten recently reported many favorable results, as did also Wolf in two cases of scarlatinous otitis affecting the labyrinth. Its employment seems rational, for with increased exudation into the various parts of the auditory apparatus, the absorption of inflammatory exudations would seem to be favored, thus relieving pressure upon nervous tissue and facilitating easy motion in the joints between the ossicles of the ear. I have not employed this remedy in a sufficient number of cases (not more than ten cases), but in this limited number the results were rather doubtful and altogether rather unsatisfactory. It is a fortunate fact that in many such cases in which the remedy seems indicated, marked improvement in hearing not infrequently occurs after two or three months even when nothing is done, so that the prognosis in these cases is indefinite.

In conclusion, I would call attention to an unexplored field of observation which would be the means of giving information having an important bearing on our knowledge of scarlatinous otitis, and one which lies within the province of the general practitioner and is inaccessible to the aurist: To what extent is the frequency of scarlatinous otitis, and to what degree is its severity influenced by the treatment of scarlatina, and especially by the treatment, both general and local, of those cases of scarlatina which are complicated by diphtheria?

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THE

PHYSIOLOGY AND PATHOLOGY

OF THE

SECOND DENTITION.

Read before the New Jersey State Dental Society, at its Annual Meeting, 1890.

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THE

PHYSIOLOGY AND PATHOLOGY

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SECOND DENTITION.

A YEAR ago I had the honor of addressing this learned Society on the subject of "The Teeth as a Factor in Diagnosis." Naturally, as a clinician, I looked at my subject from a practising physician's stand-point. The discussion, however, which my paper excited showed that the dental profession is fully alive to the economic importance of the teeth, and has noted and studied the peculiarities in these organs which are caused by different cachexiæ and various morbid states of the system, whether hereditary or acquired. Having been doubly honored by a second invitation to read a short essay before you this year, it has occurred to me that one might with profit take up another phase of our subject of last year and consider the physiology and pathology of the second dentition somewhat in detail. When we reflect that the infant comes into the world with the rudiments of four permanent teeth, we may fairly say that the physiology of the second dentition begins in intra-uterine life and terminates with the eruption of the wisdom teeth, say at twenty years of age; we have then to do with the most important third of human life, at least from a physiological point of view, and a thorough discussion of our subject would take us over a great extent of ground, much of which is already familiar to you. I will not, therefore, weary you with an elaborate discussion of the physiology of the first twenty years of life, but will endeavor to point out the relation that the process of the second dentition bears to the general health, and will supplement this with a few remarks on the hygiene of this period, which I trust will not be without interest.

Human life has roughly been divided into periods of seven years, and it is estimated that the whole body will be practically transformed by the slow process of elimination and substitution in about that period of time. The period of life from birth until the com-

pletion of the second dentition naturally divides itself into three periods of about seven years,—viz., first, until the complete eruption of the first four molars of the permanent set; second, until the complete eruption of the second four molars of the permanent set; and third, until the complete eruption of the third four molars. Of course these periods are subject to considerable variation, especially the last; but they are constant enough for the purposes of discussion, and afford convenient divisions of our subject. The first period, *i.e.*, from birth to seven years of age, is, I am inclined to assert, without fear of contradiction, from a physiological stand-point, the most important period of human life, and I believe that it can be shown that the care of the child up to seven years of age has more to do with his subsequent development than any other factor, heredity, perhaps, excepted. During this period the crowns of all the permanent teeth, except the third molars, are formed. The brain experiences over two-thirds of its complete development. The child has mastered the use of his senses, has learned to reason, has learned an elaborate and difficult language, and has often learned to read, etc. In short, in no other seven years of our earthly existence is a corresponding advance made. Now, what marks the conclusion of this period? I reply, the complete eruption of the first molar teeth of the permanent set. Owing to the varying lengths of time that different children take to reach the same stage of development, the eruption of these teeth affords a far better and safer criterion of the completion of the first physiological period of life than the mere lapse of months or years. By the English factory laws a license to work full time is not given to the youth of either sex until the second four permanent molars are completely through. This has proved to be a more reliable test of physical capacity and endurance than age or stature, since often the tallest and largest children are really the most immature and frail. Nor is it unnatural that it should be so. *Ceteris paribus*, it must take longer to develop a large body than a small one, and the old-fashioned phrase, "outgrown the strength," like most popular notions, has a foundation in fact. Naturally the development of the first four molars has less interest from a commercial point of view, but it undoubtedly marks as important a stage in bodily development as does the eruption of the second molars.

We see, then, that the complete eruption and occlusion of the first molar teeth mark a very important era in human life. The brain has attained very nearly its growth, although it increases slowly up to twenty or even thirty years of age. Its growth sub-

sequent to seven years of age is much slower than previous to that time.

The body has also at seven years of age attained about two-thirds of its height and over one-third of its average weight, if its processes have gone on normally, and I think I have said enough to show that the complete eruption of well-formed first molars is the most important criterion and sign of a normal development of these great powers and a completion of the first great division of human life.

The second physiological period is, like the first, ended by the complete eruption of four molar teeth,—viz., the second permanent molars. During this period the rudiments of education are secured, the character is for the most part formed, etc. By fourteen years of age a pretty fair estimate can be obtained of the way in which the child will turn out physically and mentally. Are there any especial features that interest us as clinicians in the second dentition, especially from the seventh to the fourteenth year?

The late Dr. James Jackson, of Boston, in his well-known "Letters to a Young Physician," dwells at some length upon the diseases of the second dentition, as does also in another place our distinguished countryman, Dr. George B. Wood. As there are those who deride the considerable importance accorded to the first dentition from a clinical stand-point, no doubt there will be very many who will assert that there is little or nothing to be said in the matter of the clinical importance of the second dentition, but I think it can be easily proved that the latter class seriously under-estimate the significance of the second as well as of the first dentition.

Mr. Jonathan Hutchinson says that the first four molar teeth are the test-teeth of a condition in children which leads to the formation of cataract, and which is also characterized by weak teeth, deficient in enamel. He says that there is no especial alteration in the form of the teeth, nor is the enamel uniformly defective and soft. It is wanting in irregular patches. There is perhaps no man living who has more carefully studied these questions of diathesis, development, and heredity than this distinguished Englishman, and the great value that he has been led to attribute to the teeth as marks and signs of constitutional vigor, or its opposite, and of normal, abnormal, or retarded development, is, to my mind at least, a marked tribute to the great importance of the line of inquiry upon which we are now engaged, and which, as I said before, has not as yet been sufficiently followed up.

Of course, all divisions of human life into periods of years are merely arbitrary. A man's earthly existence is not like a ball-player running his bases. We do not reach one period of life, then stop running and wait for another chance. We advance pretty steadily from the cradle to the grave, developing up to our prime, and then gradually deteriorating until our enfeebled bodies will no longer sustain life. Of course some advance by more rapid strides than others, just as one man may run faster than another. Some children at five are as well developed as others at seven, and I believe that it is by the eruption of the first permanent molars that we are to judge of the completion of what we may call the first great physiological period of life. If all goes normally and well, "the six-year-old molars" should appear at six years, should pierce the gums without too great constitutional disturbance, should not be soft and carious, but firm and well formed, and the child should almost be unaware of their arrival. If all this happens, we may assure the parents that the child promises to grow up healthy, and may be now sent to school. Dr. Anstie, Dr. Day, and others have pointed out the evils of sending children to school too early, and putting upon the system, already taxed nearly to its utmost, the additional strain of study, confinement, and the bad air of the school-room. No young child should be urged or driven to study, and precocious children under nine or ten years of age should be held back rather than pushed forward.

If, however, the teeth are not normally erupted as to time, and are soft and perishable; if their eruption be attended with convulsions or other constitutional disturbance, the sign is as plain as a pike-staff that the child should be immediately put under a doctor's care. It is absurd and even criminal to attribute to the visitations of Providence what we have brought upon ourselves by our ignorance and folly. I ventured to assert above that the proper care of children during the first seven years of life has more to do with their subsequent health and development than any other factor, heredity alone excepted, and this, I believe, can be proved. It has not been proved upon a large scale, because it is not as yet possible to bring up large numbers of children physiologically. When, if ever, the flattering dreams of Mr. Edward Bellamy are realized, and individual life is merged in that of the community, perhaps children can be properly brought up. At that time the doctors will perforce become communists,—because there will be so little for them to do. Nature has given us in children's teeth a sign which we cannot afford to neglect.

I think it can be easily shown that many of the morbid conditions and diseases of adolescence are due to the second dentition. In addition to the distinguished American physicians whose names I have just mentioned, I can give a number of others who attribute serious and even fatal maladies to the second dentition. J. Russell Reynolds says that there is no doubt that epilepsy may be caused by the second dentition. Erb and Heine mention teething as a cause of poliomyelitis anterior and spinal meningitis, and many other writers speak in a similar strain. Dr. Louis Starr has recently published a paper in the *Therapeutic Gazette* on the diseases of the second dentition and their treatment. Several writers, among others, Dr. Mulveany, of Brooklyn, have called attention to the association of hip-joint disease and the eruption of the first permanent molar teeth, the majority of cases of this complaint showing themselves between the fifth and seventh year. Dr. Mulveany asserted that he had never seen a case that did not begin during the period mentioned. He also gives some interesting cases of pseudo hip-disease in small children, all of them evidently too young to simulate the complaint. In one of these, so good a surgeon as Dr. Sayre is said to have been so competely deceived by the symptoms and history of the case as to have advised an exsection of the head of the femur, and actually to have cut down with the intention of removing this part of the bone, only to find that the joint was in a healthy state.

The cases all recovered readily after the complete eruption of the teeth. Dr. Starr says that the anterior molars generally give rise to more marked disturbance than do any of the teeth that replace the temporary set.

What Eustace Smith calls "mucous diseases" are more generally due to the second dentition than to any other cause or causes. I quote Professor Starr in the following description: "In this condition there is a nervous flux from the whole internal surface of the alimentary canal, which mechanically interferes with the digestion and absorption of food and greatly impedes nutrition. It may be met with at any time during the eruption of the permanent teeth. The affected child is emaciated and weak. His face is pale, though subject to marked changes in color. At times a circumscribed crimson flush appears on one or both cheeks. Again there is so much pallor, particularly about the lips, that fainting seems imminent. The eyes are surrounded by bluish circles that deepen when the face pales; the conjunctivæ are muddy and there is occasional squinting. The skin is sallow, dry, and rough, and the hair lustre-

less and faded. There is decided pallor of the oral mucous membrane. The tongue is flabby, indented by the teeth, and glazed, and the breath is heavy and fetid. The appetite in the beginning fails, then becomes capricious, and finally is almost insatiable. Eating is followed by a sensation of drowsiness and by eructations of flatus and acid liquid. Tympanitic distention of the belly and pain are always present.

"Pain may be general when it amounts to a little more than a sensation of soreness, or, more frequently, it is limited to the left hypochondrium, and then is stitch-like in character. Either variety may be constant, or present only after meals; in the former case there is temporary increase of discomfort after eating. Paroxysms of severe pain about the umbilicus, with great pallor of the face, are sometimes observed. Constipation of the bowels is usual, and the evacuations which take place at intervals of two or three days are scanty and composed of small, hard, dark-colored lumps with a quantity of mucus. By day the patient suffers from headache, is languid and ill-tempered. At night he is restless, grinds his teeth, starts from sleep in terror caused by frightful dreams, screams or laughs incoherently, and for a time seems unconscious of his surroundings. Somnambulism and nocturnal incontinence of urine are quite common.

"There is no alteration of the temperature, the pulse is feeble, and there is frequently a slight dry, hacking cough, unquestionably due to reflex irritation of the larynx. The urine is diminished during the continuance of severe pain, but is voided in large quantities at the termination of the paroxysms.

"At intervals of two or three weeks violent vomiting and purging occur. During these attacks, which last from one to three days, a large quantity of mucus is expelled. There is slight fever, and the tongue becomes less flabby, more pointed, and covered with a thick, white, feathery fur, except along the sides, where several smooth, red, glazed patches of variable size and shape with irregular indented margins appear. The clearing-out process is followed by temporary improvement, but the symptoms slowly return and grow worse to culminate in another attack. The course of mucous diseases is very protracted, extending over months, and, while there is no regular progression, the symptoms tend to grow more and more serious as time lapses.

"Hacking cough, emaciation, and debility may suggest tuberculosis. A diagnosis, however, can be readily made by noting the state of the tongue, the mucous stools, the color and condition of

the skin, the absence of pyrexia, except in the attacks of vomiting and purging, the periodicity of these attacks, the diurnal drowsiness and nocturnal terrors, and the irregularity of the course. At the same time, the outset of the symptoms after the commencement of second dentition is a point of importance.

"Mucous disease is not dangerous in itself, though by reducing the strength it predisposes to other and much more fatal diseases."

In another place Professor Starr says, "Diarrhœa is a very constant attendant upon second dentition. It is most apt to arise in the changeable weather of spring and autumn, and again the general depression produced by the coming of the second teeth would naturally favor the development of any constitutional tendency; and, having traced the connection in several instances, I have no doubt that not a few cases of tubercular ulceration of the bowels owe something to this 'etiological factor.'"

I have quoted at such length because the above is a good, even a graphic, description by an eminent clinician and writer of a common complaint of childhood which is generally referred to almost any cause but teething, as, *e.g.*, worms, the approach of puberty, malaria, etc. Dr. Jackson wrote that, since having satisfied himself of the etiological importance of the process of the second dentition, he had almost given up the use of worm-seed and other reputed vermifuges, because in his opinion most of the troubles imputed to worms were really due to something else.

A case is given of a boy of about twelve years of age who developed a chorea which lasted for several months and increased in severity; finally the lad fell into an epileptic fit. An examination of his mouth showed the gums over the second molars to be swollen and tender, and a free lancing of these finally relieved all of his symptoms. Fleiss gives a case of a small lad aged about five, who was preparing to cut his first molars; his anterior milk molars were lying half decayed in the gums, and near them were the edges of the permanent molars in a row. After a night of great feverishness, restlessness, and grinding of the teeth, the child arose in the morning with the left arm completely paralyzed. It was determined to extract all the carious teeth, in the hope of relieving the paralysis. However, the child was killed on the same day by an accidental fall upon his head. A post-mortem examination was held and showed, apart from the fracture of the skull, great congestions extending from the roots of the brachial nerves on the left side to the shoulder, neck, and face. "There appeared to be no doubt that dentition had produced this state of the veins." We

should note in passing a curious symptom and its consequences. Irritation of the gustatory branch of the fifth nerve perverts or destroys the sense of taste. This nerve, being the one principally involved in the process of dentition, is already in a state of irritation. The loss of taste takes away or transforms the appetite. The want of food or improper food may in its turn bring on chlorosis or hysteria, and indeed any of the complaints due to improper nourishment of the nerves, a condition in young women usually ascribed to anything except the teeth.

Concerning the nervous disorders of the second dentition, at the risk of wearying you I will quote again from Professor Starr's paper. He says nervous disorders, both sensory and motor, are encountered. "Headache is common; the pain is usually temporal and unilateral. It may be scattered, however, in the occipital region or in different parts of the face, and sometimes shifts suddenly from the temporal to the occipital region. It is lancinating, more or less constant, with no distinct intermission, and during its continuance there are restlessness, anorexia, a frequent hard pulse, sweating, dilatation of the pupil on the affected side, and perhaps dimness of vision, diplopia, and colored or uncolored spectra. One or more painful points can often be detected, and generally there is a hard, tender, moderately enlarged lymphatic gland in the submaxillary or cervical region.

"These attacks are due to disordered vaso-motor innervation, depending upon irritation of the sympathetic nerves and producing irregular contractions or spasms of the vessels (the temporal or occipital artery, as the case may be). The real source of irritation is to be found in the mouth. The mode of action may be twofold. First, from a swollen gum or carious tooth. The lymphatic vessels readily convey irritating matter to a neighboring lymph-gland, and the irritation here excited acts in its turn as a disturber of the sympathetic nerves, which furnish the vaso-motor supply to the carotid artery and its branches. This theory of the production of migraine has the value of the support of Lauder Brunton. The other method of production, and this I simply submit for consideration, is one of direct nervous connection, the sub-maxillary ganglion acting in the case of the lower teeth and the spheno-palatine in the upper as the medium of transfer of irritation to the vaso-motor nerves. The motor disturbances, while not so common as the sensory, are more varied. Reflex spasm and paralysis of the eyelid have been noted. More extended paralysis also occurs. Dr. Brunton says, 'Sometimes, however, paralysis occurs of a much more

extensive character, in consequence of dental irritation, especially in children. Teething is recognized by Romberg and Henoch as a frequent cause of paralysis, appearing in children without any apparent cause. According to Fleiss, paralysis of this sort occurs more commonly during the period of the second dentition, whereas convulsions generally occur during the first. Sometimes recovery is rapid, but at other times the limb atrophies and the paralysis may become associated with symptoms indicating more extensive disturbance of the spinal cord and brain, such as difficulty of breathing, asthma, palpitation, distortion of the face and squint, ending in coma and death.'"

Besides the intestinal catarrh, already noted in growing girls, catarrhs of the vaginal mucous membrane have occurred, and have led to serious charges against the purity of the patient and to much family unhappiness. Dr. Mulveany, already quoted, has given an instance in a girl of ten years, which disappeared upon *lancing the gums* and instituting the simplest detergent treatment.

The same author gives a striking instance of enlarged cervical glands due to cutting the second molar teeth and the ease with which the true cause of such affections may be overlooked. A lad of fifteen, apparently in good physical health, was brought to a dispensary with the cervical lymphatic glands in the left side of the neck enormously swollen. The surgeon who brought him desired the opinion of his colleagues as to the advisability of dissecting out the glands. After considerable discussion, the patient having been examined by the other surgeons present, Dr. Mulveany inspected the mouth carefully and found that the upper and lower second molars on the affected side had not yet protruded and were irritating the gums. The boy thereupon volunteered the information that about a year previously the glands on the other side of his neck had been similarly affected, and that he had consulted some medical man who had cured (*sic*) him.

It is hardly necessary to add that the lad escaped a difficult and dangerous operation.

Many young girls show decided hysterical symptoms previous to the eruption of the second molars which are generally attributed to the approach of puberty. To the lay mind, at least, the approach of womanhood covers a multitude of pathological sins, and the allegation of this as cause of any symptom or set of symptoms is generally quite satisfactory. Braxton Hicks has pointed out that puberty is due to a slow development rather than a sudden change. The female genitalia begin their growth *in utero*, and it is rather that their com-

plete development is announced by the amplitude and rounding of the figure than that the essential organs of womanhood spring into complete and perfect existence just at the time when the form is beautified and the menstrual function established. In other words, arrival at puberty is only the completion of a stage of human development, just as the eruption of teeth is, and generally takes place shortly after the complete eruption of the second permanent molars. Indeed, it may be retarded or rendered painful and difficult by interference with dentition.

It is certain that cases of hysteria, chorea, and even epilepsy, are cured by lancing enlarged and tumefied gums over advancing permanent teeth, and the assumption that the difficult or retarded menstruation is due to the disordered state of the system caused by dentition is more in accordance with acknowledged pathological facts than that the disordered state of the system is caused by the delayed menstruation.

Dr. Garretson remarks, in his Oral Surgery, that while dentition is a physiological process, still it is like that of utero-gestation, one of continuous irritation. While I readily admit that the disorders and disturbances of teething are not solely due to the formation and eruption of the teeth, and I believe them to be chiefly due to the perfecting of important physiological changes in the brain, stomach, glandular and osseous tissues, going on simultaneously with the eruption of the teeth, I assert, without fear of contradiction, that the normal and peaceful eruption of healthy teeth is our best visible criterion of the progress of these processes and of the healthfulness of the system, and, on the other hand, delayed and irregular eruptions of the teeth as surely evince some constitutional vice or evil hygienic condition.

Dr. Mulveany, from whom I have already largely quoted, cites a case of a young woman fifteen years of age, who had suffered for years with a catarrhal ophthalmia which finally readily yielded to treatment after twenty-eight permanent teeth had been cut, to be followed in a fortnight by a copious, offensive purulent discharge from the vagina, accompanied by excoriations from the mons veneris to the anus, which naturally led to suspicions of unchastity. However, the simplest treatment cured the condition in a few days and the girl menstruated for the first time. Here was a case of catarrhal diathesis with local manifestations in the conjunctivæ kept up by the irritation of dentition, which persisted in spite of treatment until after the eruption of the second molars. The dyscrasia then showed itself in another region, provoked by the unusual activity

of the uterus (about, one might say, to enter upon its life business); after the function of this latter organ had been established the vaginal catarrh disappeared.

It is doubtless true that many morbid symptoms really due to dentition are falsely ascribed to the approach of puberty in both sexes. It is not asserted that the former is all-important, or even more so than the latter. It is only claimed that as a factor in the production of the diseases of childhood and adolescence dentition has not received due recognition.

Any unused function is gradually lost. We use all our muscles too little; our chests are narrow and our lung expansion small because we do not exercise our arms and our lungs enough. Our teeth are poor and our jaws narrow because we do not chew enough. Ambrose Paré spoke of a "rusty filth which attacked and destroyed the teeth and was chiefly due to the omittings of their proper duty,—that is, chewing."

I asserted in the early part of this paper that proper care and food in the first seven years of life would in my opinion have more influence upon subsequent development than any other factors whatever, heredity alone excepted. Perhaps the statement was somewhat extravagant. Let us consider it. Of course, by proper care, I meant proper medical supervision of the child and a thorough knowledge of the child's family history, so that the best measures might be taken to ward off the hereditary evils, which, like the evil spirits of which we read, are in constant attendance upon the helpless children, waiting to pounce upon them just as soon as some weakness or injury shall give the opportunity. Syphilis and tuberculosis leave their impress upon the children unto the third and fourth generation, but these tendencies can be successfully combated, even in the first, and it is our duty to study constantly all the conditions and all the signs of dyscrasiæ, of development, and of diathesis, that we may know what we have to combat and may have our weapons ready and burnished. The demons of disease and ill health have fastened themselves pretty strongly upon unfortunate humanity, and the best-directed efforts are feeble enough against them. But that does not excuse us from the eternal vigilance which is the price of nearly everything worth having in this life. I think that it is Professor Peirce who has pointed out that the teeth can be altered and developed by proper exercise, care, and food. He alludes to the fact that the larvæ of the honey-bee are developed into a queen bee or into working bees according to the nature of the food they receive. As to practical suggestions, I

have purposely avoided discussions of the treatment of particular diseases as I have gone along, fearing to make my paper too long, and because the treatment of special conditions is unimportant compared to the hygiene of child-life. Give a young child twelve hours sleep, plenty of sunlight, good air, and proper food containing plenty of phosphates, and he will develop that reserve of nerve-force which is needed to carry him through the emergencies of development.

Of the third period of our division a very great deal can be said. The hardships and vicissitudes experienced in getting the wisdom teeth and the general worthlessness of the product have engaged abler pens than mine, and I take it for granted that you are all more familiar with this part of our subject than with what has gone before, inasmuch as people suffering with their wisdom teeth are more apt to consult the dentist than the children and youth of whom we have just spoken; and also because of the more obvious and immediate connection of various troubles and the eruption or non-eruption of the wisdom teeth. However, there are certain physiological facts connected with this process which are not so generally known and which may profitably take up our time for a few moments longer.

There are many cases of eczema and other skin-disease, for example, which attack the young and which are often incurable until after the eruption of the third molars. You are all familiar with the acne of young people, which, commonly speaking, gets better and worse again during a period of years, but is finally cured after the completion of the second dentition. The connection of eczema with the eruption of the teeth is, like tooth-rash in the first dentition, more obvious, and it is probably more tractable to treatment when the irritation of teething is over than the acne.

The nervous diseases connected with the cutting of the wisdom teeth are even more pronounced than those that come earlier in life, and the reflex neuroses are at this time more marked. You are all probably familiar with some of the numerous recorded cases of complete blindness of one or both eyes, of deafness, of spasms, migraine, chorea, epilepsy, and insanity due to impacted wisdom teeth. Some of the functional disorders connected with the wisdom teeth are perhaps not so familiar. Dr. Anstie mentions a case of severe uterine neuralgia clearly found to be due to a carious tooth, which was not at all sensitive. A case of alarming uterine colic in child-bed was, after ineffectual treatment, found to be due to an unerupted wisdom tooth, and was cured by the free use of the lancet over this tooth.

A curious case of vesical irritability is recorded in which the patient experienced pain in the teeth upon assuming the erect posture, especially when the feet touched the floor on arising in the morning.

Dr. Garretson gives a case of severe pain in a lower bicuspid tooth which was not relieved by extraction of the tooth, but was finally cured by curing an erosion on the inner surface of the fundus of the uterus. Hundreds of similar instances of reflex symptoms have been recorded. Dr. Mulveany says that he was frequently consulted by anxious young husbands because their youthful partners did not conceive. He always assured them that when their wives got through their teething they would have children, but that they were not likely to become mothers until quite over the infirmities of childhood.

I think that enough has been said to show that the eruption of the permanent teeth is by no means an unimportant process so far as it affects the health. Dr. Kingsley says that a perfect dental development is the result of well-balanced physical and nervous systems, without hereditary taint. "Abnormalities of development are due to disturbance of the trigeminal nerve during the period of the formation of the crowns of the permanent teeth. Neither lunacy nor insanity can have any direct bearing upon the development of the teeth, but would be most potent for evil if transmitted." The doctor continues:

"I do not hesitate to place it upon record that the next generation will see more of abnormality in dental development and an increase of nervous and cerebral diseases, and that the two are correlated and spring from the same cause, are not, strictly speaking, symptomatic of each other, but are associated and frequently have a common origin."

As dental irregularity is often accompanied by intellectual precocity, it may be said to be a sign of nervous excess in such persons.

As we glance backward over our subject we observe that the teeth of the permanent set which give the most trouble as they come through are those which do not replace milk teeth, *i.e.*, the permanent molar teeth. Their eruption may then be said to resemble the eruption of the milk teeth, and so it does. Convulsions, diarrhœas, catarrhal diseases, etc., are more apt to show themselves with these than with any other of the permanent teeth. Of these teeth, also, it may be fairly said that they resemble milk teeth in being of softer consistency and more susceptible to caries than the rest of the permanent set. This is surely true of the first and third molars.

Many theories have been advanced to account for the perishable character of the first and third permanent molars. Perhaps the first are poor because they are erupted so early, some claiming that they should not strictly be called permanent teeth, that they are a sort of link between the two sets, and that they should be extracted in time to make room for the third molars, which, it is claimed, would be good teeth if they had a chance to come through the gums promptly and easily, and could be kept clear and free from crowding.

This is a pretty theory, but scarcely a tenable one. Our jaws are small, our teeth poor and crowded, chiefly because we eat too highly-prepared foods, and do not exercise ourselves enough in chewing.

Our growth should be ever peacefnl and regular. This constant talk about over-strain, neurasthenia, etc., only means that some one has sinned, either this man or his parents, and I judge it to be generally the latter, that he (or she) is born with "nerves." If we lay the foundation well for our children and teach them to avoid our mistakes in matters of hygiene, we shall confer upon them benefits that wealth or learning cannot give. What I am constantly protesting against is the one-sided view of education that Americans entertain. We must educate—*i.e.*, draw out—the body and the mind; we must develop the chest, the arms, the backs, the jaws and teeth of our children, if we wish book-learning to be of any value to them or wish them to get any permanent good out of life or be of use in their day and generation.

Physical laws are God's laws, and if they are disregarded the punishment is swift and sure.

However, I am not one who takes a gloomy view of the physical future of the American people. I understood Professor Truman to say in your meeting last year that the teeth of our people are constantly growing better. I believe this to be true, and I believe that to our excellent and conscientious dentists much of the credit for this improvement is due.

When the laws of health are better understood, when the signs of a feeble constitution are more quickly read, when our people learn that building up and developing a normal body is really a simple thing, the Americans will become the finest race on the face of the earth.

DISCUSSION ON DR. R. C. NEWTON'S PAPER.

Dr. B. F. Luckey.—Mr. President and Gentlemen,—When I read on the programme that I was to open the discussion on this paper

I was astonished, not having been forewarned and not having had an opportunity to read the paper since.

I do not think it would have made any difference if I had had opportunity to do so, for I do not see, upon listening to it, that there is anything in it that is open to discussion to any extent. As a dental paper from a medical source it is one worthy of commendation. As a general thing, such papers coming from medical sources are very apt to be loaded and surcharged with ideas that are fallacious. As a rule, medical men give so little attention and thought to dental subjects that when they do consider them they do so a good deal in the manner of the laity. The doctor has dealt to-day with the subject of "The Physiology and Hygiene of the Second Dentition," and it is a compilation of well-known facts that have been gathered apparently from reliable sources. But, so far as I remember, none of his remarks bring out anything entirely new, and he reduces the work of a dentist down to the use of a lancet. Almost all of the troubles he mentioned occur through retarded dentition, or irritation from retarded dentition. I think, perhaps, I can do nothing more interesting than to cite a case which has just recently passed through my hands to illustrate that if physicians were a little more conscientious in consulting with their brotherhood, the dental profession, they would not only confer a boon upon their patients, but also relieve themselves of a good deal of the odium which attaches to the mistakes which all professional men are liable to make.

A patient, probably thirty years of age, presented a very badly-swollen jaw. The swelling was of such a nature that the patient was unable to open his jaws sufficiently to permit of a proper examination of the whole of the mouth. He had been under the treatment of a physician for nearly three weeks, and was nearly exhausted from pain, suffering, etc. His physician told him there was dead bone back of it, that there was necrosis, and he would have to operate to relieve him. He came to see me about it. I could not with the little chance I had for exploration determine satisfactorily to my own mind what the trouble was, but I came almost positively to the conclusion that it was due to a wisdom tooth coming through in such a direction that it was not possible for it to get into position: it was tilted over in the ramus of the jaw and came directly against the distal surface of the second molar tooth. I informed the gentleman that, as far as I could judge, that was his trouble, that if he would open this abscess and allow it to discharge, and come and see me as soon as he could open his

mouth properly, I might be able to help him. The abscess was opened and discharged freely, but the hard swelling did not subside, and the man was not able to open his mouth for two or three days. He suffered a great deal, and I advised him again and urged him not to poultice from the outside, his physician constantly recommending that process. I explained to him it would not relieve him, but the probability was he would have a scar on the outside of his face as long as he lived. I informed him after the second visit that I was sure there was no necrosis, and our only hope was to extract the second molar, which was perfectly sound, and allow the wisdom tooth to come in in that way. He did not feel willing to agree to that, and of course we could not perform any operation until his jaws could be opened. He came to me the second time some weeks after and I gave him the same advice, but he did not feel inclined to accept it; he said he would come when he could open his mouth; in the mean time he went back to his physician and made an appointment; they anæsthetized him, opened his face, went for the dead bone, and did not find it. The man recovered after awhile sufficiently to open his mouth and came to see me again; by that time the wisdom tooth had begun to show itself slightly, and I was satisfied thoroughly then of the diagnosis, and again advised him to submit to the operation; he did so and recovered entirely; but he has two horrible scars on the outside of his face which he will carry to his grave. There was a case of suffering for some two months or more, which could have been cured at the start, in all probability, had there been a consultation between the physician and the dentist.

As to the hygienic portion of the paper, I do not see that I can add anything to what the doctor has said as to the care and nutrition of the patient, excepting to advocate the constant and thorough use of the teeth and their cleansing. The teeth, like all other organs of the body, require use to get them in their proper condition. The selection of soft food and the avoidance of the use of the teeth creates a tendency, as similar causes do in all other organs, to degeneration, and although abrasion may be somewhat wearing on them, it still has a tendency to the hardening of the teeth.

Professor C. N. Peirce.—I speak because I wish to congratulate the New Jersey Society on having a physician read a paper before it, and I want to congratulate the author of the paper on the arrangement of his subject and his treatment of it, because I believe it is a very great step in advance of a large class of medical practitioners. The reading of the paper has given me a good deal of pleasure, and although perhaps he has not illustrated anything but

what many of our leading dentists have been familiar with for years, yet it shows that the medical profession are recognizing the influence of the teeth in certain directions. It also adds greatly to the status of the dental profession in the eyes of the medical profession when it is recognized that the teeth themselves are capable of producing such systemic disturbances. I could make no criticism at all of the paper excepting as to a remark that the character of the first molars and the influence of dentition was quite as great in the development of a child through youth and to manhood or womanhood as hereditary influences are. While I recognize the influence of dentition very fully, yet I want to say that hereditary conditions are in advance of the results accomplished by individuals. There may be temporary influences during the period of dentition that will effect the development of the teeth in an infant, and will necessarily cause great disturbances in the eruption of the permanent teeth. As the doctor is well aware, when a child is born it has the twenty deciduous teeth largely calcified; those teeth are influenced by the action of the health of the mother during the period of gestation. During the first year of life we have the incisors and first molars undergoing calcification. The first molars at birth often have the point of the cusps already completed. The health of the mother must necessarily produce imperfect development in those first molars if it is bad, and unless that child has peculiar influences about it during the first two years of its life all the permanent teeth will be imperfectly calcified and oftentimes produce great disturbance in their eruption in consequence of the imperfect nourishment of the infant.

Otherwise, I have to say amen to every word of the paper, with just that little criticism.

Dr. J. A. Osmun.—I have nothing to add excepting words of commendation for the paper, and as I am delighted with it I can only re-echo the sentiment of Dr. Peirce,—that dentists and physicians are becoming brothers in a common cause, and the time is coming, I hope, when they will stand shoulder to shoulder in their efforts to relieve the sufferings of their patients. It should be so.

The doctor's paper points out in a broad way the influences that come from first and second dentition. From a physician's standpoint he deals with the body as an entirety, but he also feels that he should have the benefit of consultation with his brethren who make a study of this particular portion of the body. I say it is a happy omen of the time when we shall stand shoulder to shoulder in our efforts to alleviate pain.

Professor Charles Mayr.—Professor Peirce's remarks have suggested to me a most interesting subject, namely, the question of how the various cells in the child during the period of gestation are connected with the corresponding cells in the mother. You know there are two theories, one is that from every tooth in the mother's mouth there goes a nerve-filament to the growing tooth of the child, and I think the observation is not uncommon that women about that time feel various organs getting out of order; first the stomach is wrong, and this is wrong and the other is wrong, and then the teeth are wrong. I think that is the first shock of the connection, and afterwards it is normal. We can remember our experiences when we were first thrown in the water to learn to swim; we remember it all our life long, but afterwards we tumble in a dozen times and forget all about our sensations, so that I think the moment a woman in that condition feels her teeth aching the connection is made and the teeth begin to calcify, and the child too has to get its starting-point from the lime salts of its mother's teeth.

I believe myself too much in the individuality of atoms; in my opinion there are no two atoms exactly alike, every atom has its individuality.

The other theory is that no such connection exists, but an impulse of wave motion proceeds from the teeth of the mother towards the child. Which of the two theories is correct is hard to tell.

Dr. Sanger.—Suppose the mother's teeth are false, what then?

Professor Mayr.—Well, of course the nerves of the teeth are still there, the teeth and the endings and all that; the nerve-endings which formed the teeth before are still there unless the whole tooth and nerve is destroyed.

Dr. Walker.—Does Professor Mayr think that the lime-salts are gradually absorbed from the bony system of the mother and pass through the circulation to the child?

Professor Mayr.—As I said, there are two theories, one being that only the wave motion is transmitted, and the other that entire particles from the part are transmitted down to the corresponding part in the child.

Professor Peirce.—There are a great many mothers who bear children without any detriment to the teeth. The mouth is kept in a perfectly healthy condition all through the period of gestation, and not only one, but sometimes ten or twelve children are borne, and the teeth are always in good condition. On the other hand, we find mothers who become exhausted and are not able to pro-

duce sufficient nutrition to build up the embryo, and so the deficiency arises through the inefficiency of the mother.

I know of an instance where, in the case of an infant, the original teeth were entirely deficient of enamel. The permanent teeth came through in no better condition, and the difficulty was that the mother had just that trouble during the period of gestation. The child never had good teeth because, during the period of gestation, there was no ability to build up those structures.

Dr. R. C. Newton.—Our indiscretions sometimes serve us well. I think I read that line concerning hereditary wrong, but it drew forth a very interesting talk from Professor Peirce. I think it all goes to show the main contention of the paper is right,—that an immense amount of good can be done if you treat those matters intelligently and at the right time. Nine times out of ten it seems to me the constitution is good enough if the people having the care of children knew what was needed. Doctors have not given enough attention to the teeth; they do not care enough about it. Until within a few years they were brought up to consider the body as a receptacle for drugs which should remedy existing evil. The whole tendency of modern thought is to prevent the evil rather than to go upon the idea that for all troubles there is a remedy. As Professor Mayr has said, when we are able to restore decay we shall have discovered the elixir of life. None of us believe we shall ever find the elixir of life, but a little elixir of common sense in the bringing up of children would be a very good thing.

Dr. J. A. Osmun.—That is the idea I tried to bring out yesterday. The public ought to be better educated, and the province of dental and medical societies is to disseminate more knowledge among the laity.

Dr. Newton.—That is the view I have always taken, and for that reason I have always been willing to do all I could in that direction.

The Etiology of Empyema in Children:

An Experimental and Clinical Study.

BY

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The first aspiration and exploratory puncture of the pleural cavity was performed by Bowditch, the American physiologist, and from this bold innovation upon the former methods in vogue may be traced our decided advances in the treatment and proper understanding clinically of the pathological conditions of the pleura. In his address before the Tenth International Medical Congress, Sir Joseph Lister in speaking upon empyema said: "There are few more beautiful things in antiseptic surgery, as contrasted with the results of former practice, than to see the abundant purulent contents of the pleural cavity give place at once to a serous effusion, rapidly diminishing from day to day." Surgery has therefore been in advance of pathology, and it was reserved to a very recent period for some enlightenment upon the nature of empyema and pleurisy to appear, and supplement the surgical advance in this department of medical learning.

This advance has been made through the avenues of modern bacteriological methods. I am certain that with the light thus thrown upon the nature of empyema in the adult, the diagnosis and understanding of this disease will have received incalculable aid, and the future treatment in both surgical and medical directions will be based upon more certain data than hitherto. I think we have passed that stage which justifies the general grouping of all suppurating pleural processes as empyema without modifying this term so as to point out at once the etiology. If the above is true of the adult, we would naturally expect the same phase of this question when applied to children. Though we have many of us taken for granted that what was true of the adult might be equally so of the infant and child, it has been the object of the author of this paper to make such an inquiry in exact channels, as had been done by others concerning the adult. The literature upon the etiology of empyema in the adult is not so very extensive, if we regard those works only which are really systematic, and therefore of satisfactory merit. The first complete contribution, an attempt to formulate the different varieties of

empyema from a bacteriological standpoint, was that of A. Fränkel.[1] This paper contains the result of a series of studies extending back as far as 1886. The author divides empyema into the following groups:

Those in which the etiology is still a matter of speculation. In these we cannot point to anything positive, because both the clinical history and bacterioscopic examination of the purulent exudate give no support to any definite theory. In these cases the exudate upon examination fails to yield anything but a microörganism which is found in processes of diverse nature in the body. From the chain-coccus, or streptococcus pyogenes, we could with much justice presuppose an antecedent pneumonia; we might follow this chain-coccus in its migrations through the lymph-channels of the pulmonary pleura; but, on the other hand, there are authors who, like Fraentzel, believe in the occurrence of a "pleuritis acutissima." Unfortunately, this disease described by Fraentzel is of most rare occurrence and doubtful etiology. Traumatism or cold have been invoked in these cases as predisposing causes which allow such microörganisms as streptococci to act upon the economy by reducing its resistant vitality. We must here presuppose the continued presence of microörganisms in the subpleural tissue, even in subjects who clinically cannot be said to be suffering from any active disease. In the course of investigations upon empyema, the question has arisen whether an effusion into the pleural cavity might from the very onset be purulent; those of experience answer in the affirmative. In children especially we are apt to think that an effusion is serous, or rather non-purulent, if the exploratory needle gives what to the eye appears a clear, not turbid fluid. The inquiries into the pathology of these cases show that those acute cases showing a serous exudate which subsequently becomes purulent are really at the time purulent (from the beginning, in the most modern acceptation). To return to our first premise, though Weichselbaum has distinctly shown, in lobar and lobular pneumonia of the adult, that the streptococcus pyogenes and also the staphylococcus are present as so-called mixed infections, yet when we come to consider the exclusive presence in the exudate of empyema of these microörganisms there is a gap to be explained which, as far as we know, has not yet been clearly elucidated.

In another group of cases Fränkel found the exclusive presence of the pneumococcus or diplococcus pneumoniæ; this microörganism was found in pure culture in the exudate derived from the pleural cavity. The pus was of a thick, adhesive character, and the cases of this group are classed as post-pneumonic or concomitant with pneumonia (lobar pneumonia). Fränkel maintains that in these cases suppuration is

[1] "Ueber die bakterioscopische Untersuchung eitrige Ergüsse," Charité Annalen, 1888, p. 147.

maintained in the presence of a closed pleural cavity by the presence of bloodvessels with the existence of a different nutritive medium than in the artificial structures.

The third group of empyemas are those whose nature is tubercular. In all these cases he could not establish the presence of tubercle bacilli, for some baffled all the aids to diagnosis of this microörganism, both in plain stain, culture, and experiment. Fränkel has come to the conclusion that the absence of any result as far as stain and culture were concerned, in the study of exudates, pointed very strongly toward a tubercular element in the etiology of the empyema. Garré and Rosenbach in experimenting with the pus of cold abscesses had a similar result. Fränkel found tubercle bacilli by staining methods in only one case of four investigated; in the others the results were negative.

There is, according to this author, a fourth group of cases, in which we can find a focus of infection situated outside of the pleural cavity. He had two such cases, in both of which the chain-coccus was found, and one of which followed a perforating peritonitis, the other a retro-pharyngeal abscess. The conclusions of this author relating to empyemas are that the presence of the chain-coccus or streptococci, or staphylococcus pyogenes is not diagnostic. The presence of the diplococcus pneumoniæ points to a preceding pneumonia or complicating pneumonia. An exudate which fails to yield any positive result either by stain or culture is in all probability tubercular.

Weichselbaum's name is also indissolubly connected with much good work in this field. He examined eleven cases of pleuritis.[1] Two of these cases were of a purulent and one of a sero-purulent nature, the remaining eight were serous. In the empyemas he found the streptococcus pneumoniæ, or what we may call streptococcus pyogenes, and in another case, which proved fatal, the diplococcus pneumoniæ was found. About this time, Fränkel published[2] two cases of empyema following pneumonia, in which he found the diplococcus pneumoniæ.

The above historical review would be incomplete without the mention of the important work of Ehrlich in the study of pleurisies.[3] He investigated by staining methods the bacterial character of fluids obtained from the pleural cavity in forty-five cases of pleurisy; of these, nine were tubercular, twenty simple pleurisy, six carcinomatous, and nine empyemas of various kinds. Of nine tubercular cases in which the sputa of the patients contained tubercle bacilli this microörganism was found in only two. The negative result in the remaining seven does not by any means prove the absence of tuberculosis of the pleura.

[1] "Ueber die Etiologie der acuten Lungen- und Rippenfellentzündungen," Medizin. Jahrbücher, 1886, Heft 8, p. 483.

[2] Zeitschr. für klin. Med., 1886.

[3] Beiträge zur Ætiologie pleurit. Ergüsse," Charité Annalen, 1882, p. 207.

The work upon empyema in children is, in contrast to what has been recorded of the adult, limited to a few scattered notices in the literature. The most important is found in the work of Rosenbach, who examined the exudate of a case of empyema and found the micrococcus tenuis (pyogenes), or what is now thought might have been the diplococcus pneumoniæ, present.

The recent articles of Von Ziemssen ("Vorträge") and Liebermeister are simple literary *résumés* upon the subject.

ORIGINAL INVESTIGATION.—My own work upon empyema was framed upon the lines outlined in the above review. An attempt was made to see how far the results in children, from a bacteriological standpoint, would correspond to those attained in the adult, or whether they would differ. In June of last year, I reported in brief my results in twelve cases; there are three additional cases to report, in one of which at least some interesting points will arise. The work done was upon material which at the time, for the most part, was in a hospital ward, evidently an advantage. The mode of bacterial study was in accord with what is practised by the Koch school of investigators. Some of my cases were taken from my dispensary class; others from private practice or those of my friends. It being established both from the clinical history and physical signs that there was a probable existence of empyema, the chest of the patient was carefully cleansed with soap and water, and then with sublimate; the excess of antiseptic fluid having been removed, a clean and sterilized hypodermic needle was introduced into the chest. To avoid contamination, a new syringe was used for each case, for it is very difficult to be certain as to the cleanliness of a syringe which has been once used for pus. The ordinary hypodermic syringe was used after being cleansed with sublimate, alcohol and ether, and then sterilized distilled water. The needle was sterilized with heat in the dry oven at a temperature of 160 to 170° C., kept in a sterile test-tube and only put upon the syringe at the last moment. A specimen of the contents of the pleural cavity once withdrawn was placed in an empty sterilized test-tube and taken as soon as possible to the laboratory and examined. The examination included not only the study of the gross specimen, the preparation of crude pus upon cover-glasses in the ordinary way, and then stainings of the crude pus, but plates and cultures were made upon media to establish the bacteriological character of the fluids withdrawn. The media used for culture experiments were bouillons, gelatin, glycerin agar, agar-agar, Weichselbaum's agar;[1] blood-serum and potato plates were first made to obtain colonies, as also test-tubes prepared by rubbing the fluid to be examined upon the obliquely solidified media, as

[1] By Weichselbaum's agar is meant the agar recommended by this author, containing 1½ *per* cent. of agar, peptone, and grape sugar, with ½ per cent. of salt.

agar (Weichselbaum); and pure cultures were also obtained from the plates, and if growth occurred in the tubes, plates were made to test the purity of the growths. Re-inoculations upon other media were made; when pure cultures were obtained, these were injected into animals for experiment. The animals used were rabbits, guinea-pigs, rats and mice. The most uniform results were attained from the rabbit experiments. The microörganisms were taken from a pure culture from an agar tube and suspended in sterilized distilled water, or, mostly, a pure bouillon culture was directly injected. The injections upon animals and autopsies were performed with the ordinary precautions followed in the bacteriological laboratory.

The characters of the crude pus withdrawn from the various cases are of interest when considered in relation to one series of cases, namely, those in which the diplococcus pneumoniæ of Fränkel and Weichselbaum was present. In some of this set of cases, the pus seemed of a rather glutinous adhesive nature; it would adhere to the sides of the test-tube when the same was inclined; this pus was either creamy-white in color, or greenish, or greenish-yellow. In these cases the chest was sometimes filled with enormous clots of fibrin, which were expelled from the opening made by resection of the rib. The pus also in some cases separated in a serous and opaque greenish layer upon standing; in other cases, as in greenish pus, the same had no adhesiveness. I mention these facts, because Fränkel has pointed out the adhesive or glutinous nature of the pus withdrawn from the adult cases, and the same characters in similar cases were easily established in children. In none was a fœtid odor perceptible, either in the pus derived from the cases in which the diplococcus pneumoniæ was present, or in those in which the streptococcus or staphylococcus pyogenes was found. Primarily, none in the series I have examined were fetid. In two cases the pus became fetid after the chest had been opened, and these cases, both of diverse nature, I will attempt to explain later. In all cases the crude pus was spread upon cover-glasses and stains made both for tubercle bacilli, diplococcus pneumoniæ, and other microörganisms; care was taken to follow out a routine in each case by which tubercle bacilli (Ehrlich's stain) could be excluded. In those cases in which the capsule cocci were found by simple stains with methyl or gentian-violet, the resistance to Gram's discolorization was tested.

For the sake of simplicity, we can formulate results in the following manner: In the pus of the fourteen recorded cases, I found the following microörganisms:

The streptococcus pyogenes.

The staphylococcus pyogenes aureus.

The diplococcus pneumoniæ (Fränkel and Weichselbaum).

The tubercle bacillus (Koch).

In few of the cases did I find these microörganisms associated, but usually existing alone in a specimen of pus. The tubercle bacilli were found associated with the streptococcus, as I shall later explain. It was certainly striking, at least in my series of cases, that the organism found existed in the fluid withdrawn from the chest in so-called pure state. The pus was really equivalent to a pure culture of one of the microorganisms. It would be tiresome and of little value to go into detailed description of the staphylococcus pyogenes aureus and streptococcus pyogenes found in my cases, and it will suffice to say that they are in culture media, and when injected experimentally acted in a way exactly corresponding to all that is known of these microörganisms.

The Diplococcus Pneumoniæ (Fränkel and Weichselbaum).—When the pus from an empyema is rubbed upon the surface of an obliquely solidified tube of agar-agar of Weichselbaum, and placed in a temperature of 35° to 37° C., there appears within about six hours a thin, almost imperceptible coating very much like dew on the surface of the agar; within twenty-four hours this is more marked, though it is still very delicate and veil-like, and does not seem to grow vigorously. In the depth of the agar, if the same has been inoculated, there is a delicate veil-like reticulum. If such a surface culture be examined after successive re-inoculations, it is made up of very minute transparent punctate areas or colonies; as a whole, the growth does not attract the eye by any particular hue. If a puncture culture, as it is called, is made on agar, the area around the top of the puncture is very minute and scarcely perceptible. Colonies from pus sown upon agar-agar plates are at first so small as to be discovered only by the aid of a lens; they never attain a very large size, and are situated in the depth of the medium, and inoculated by means of the platinum point only with the greatest difficulty. The reason of this seems to be that the microörganism is so delicate that not enough adheres to the platinum tip to favor growth in the test-tube. The colonies after forty-eight hours are round, granular, with a darker centre, some of them, than periphery; they are of a transparent very light grayish tint or straw color by transmitted light; when the plate with colonies is held against a dark background, the reflected impression is that the colonies have a whitish tint. Unless this microörganism is transferred very early during the first few days to other tubes, it stops at a certain point and ceases growing—in other words, it may be lost. The colonies, especially in four or five days, begin to die out, becoming fainter to the lens and taking on the color more and more of the surrounding agar. The above applies to the diplococcus in pure culture derived from pleuritic fluid. After inoculation into animals, the growth, though the same in all essentials, seems to have attained a greater vigor, though even here, if not saved by repeated re-inoculations upon media, it dies out quickly.

Gelatin.—In gelatin, at the ordinary room temperature, there is no growth; at the temperature of 23° or 24° C. there is a very delicate growth which never becomes vigorous; along the puncture there is a granular minute beaded structure, but of the greatest delicacy, and the gelatin is not fluidified.

Blood-serum.—The growth here is much the same as on agar-agar of Weichselbaum, though the colonies making up the surface culture may be said to be more grayish in tint, or slightly more perceptible than upon the agar.

Bouillon.—Here there is first a general clouding of the medium, and then a deposit of flocculi along the sides of the tube, and finally a small deposit in the bottom of the test-tube, leaving the fluid or bouillon above finally clear. When shaken, there is great turbidity and abundance of fine flocculi.

Potato.—There is here no perceptible growth.

Ordinary agar.—It is certainly a waste of energy to attempt to cultivate the diplococcus successfully upon ordinary agar, for failure will be the most frequent reward. In other words, much valuable time has been wasted by others as by myself in making such attempts. The least variation in moisture or reaction will compromise the growth, so that I have worked mostly with the agar-agar of Weichselbaum, rather than with that made up by the old formula.

Glycerin agar—six per cent. to eight per cent.—did not seem to present any advantages. The growth was very slow and difficult of re-inoculation, and capricious.

Stainings.—The crude pus shows us the diplococcus with its capsule. I have had no difficulty in establishing the presence of the capsule coccus by means of the simple methyl-violet or gentian-violet and aniline water stain, taking care not to over-stain. If the specimen should have been over-stained, it can be easily decolorized (but this very lightly) with dilute alcohol. The pure culture shows the diplococcus from agar very beautifully, as oval or round diplococcus forms of apparently the size seen in the crude specimen without any capsule; the diplococci may be single or in chains of two or three pairs. By the Gram stain there are also seen both in the crude pus and crude culture some of the peculiar lancet-shaped forms; while I could find here and there a beautiful lancet form, there were many which upon analysis appeared peculiarly crenated, perhaps due to either unequal decolorization or overheating. The capsules in the crude specimens are decolorized by the Gram method, though the light decolorized zone of their presence may be seen by strong and favorable illumination in some cases.

THE STAPHYLOCOCCUS PYOGENES AUREUS which was met in these empyema cases could in no way be distinguished from the staphylococcus which I separated and cultivated for comparison from the ordi-

nary furuncle of the skin. It reacted in the same manner in the various culture media. In the gelatin it grew at the ordinary room temperature along the puncture and had the same yellow-gray or straw-color beaded look. The gelatin, after the third or fourth day, became liquefied; this began at the top and proceeded along the puncture in length and breadth. After a time the liquefied gelatin showed the finely granular suspended colonies, and the sediment at the bottom of the liquefied gelatin took on the same orange-yellow color.

Agar-agar.—Upon obliquely solidified agar there was at a raised temperature a very vigorous growth on the surface, and in the depth, which, at first whitish-yellow, after a time assumed a rich orange-yellow tint; the surface of the growth was moist, and its edges sinuous and raised very perceptibly above the surface.

Potato.—The well-known orange-yellow moist growth was perceptible within twenty-four hours, increasing in vigor and luxuriance; it was at first golden-yellow, then deeper orange-yellow, having the peculiar odor of the ordinary culture of staphylococcus.

THE STREPTOCOCCUS which I have isolated from cases of empyema reacts in media exactly similarly to the ordinary streptococcus pyogenes. In agar-agar plates the colonies remain small but some large, some irregularly round, others oval of a brownish-olive tint and granular appearance. In punctures the finely beaded dots and zone surrounding the top of the puncture exhibit no tendency to spread to any extent; on the surface of obliquely solidified agar a pearly-gray growth is seen (Weichselbaum's agar) made up of drop-like masses in the depth; the same beaded appearance in either a band or puncture that is seen in ordinary agar.

Gelatin.—Small finely granular colonies, at first straw-tint and sharply round, later more brownish with an olive tendency. At border, through gelatin, they appear straw-color, on surface of gelatin they have a distinct cupola (raised); they grow in depths also. In punctures, finely granular, no liquefaction of gelatin.

Bouillon.—In twenty-four hours we have a cloudy appearance, and deposit of masses on sides of test-tube, finely granular in the centre. Stainings show exquisite chains.

Potato.—Nothing characteristic or perceptible.

EXPERIMENTAL.—The experimental part of my work was laid out upon very simple lines, and is intended merely to add confirmatory evidence upon the nature of the microörganisms which I have isolated from the various cases of empyema reported. The results attained, it will be seen, are almost identical with those obtained by other observers (Fränkel and Weichselbaum).

My method consisted first, in isolating microörganisms so as to obtain a pure culture of each variety, and then injecting this pure culture into the animal to be experimented upon. The animals used were mostly

rabbits, but a few scattering experiments were performed with guinea-pigs, rats, and mice. The injections were made with sterilized hypodermic needles underneath the skin and into the pleural cavity or lung. There were no inhalation experiments. A pure culture of any micro-organism having been injected into the animal, the same was observed, and if the animal survived, it was, after a sufficient period had elapsed, killed and pathological effects noted. If the animal died from the effects, the blood, and the pleuritic, pericardial and peritoneal fluids were examined, and the nature of the contained microörganisms established in exactly the same manner as had been done with the original pus obtained from the empyema. Re-injections of these pure cultures from animals were made, as also injection of pleuritic and peritoneal fluids from animal to animal. Pus in crude state from empyemas was also injected into animals, and the microörganisms cultivated from the blood and fluids of such animals.

The most interesting series of experiments in their results were those performed with the pure cultures of the diplococcus pneumoniæ. In every case the isolated microörganism was injected into a set of animals, and the results have been noted. After the first effects of the injection had passed off (chest injections into pleura), the animal in some cases seemed to be as well as ever, but after a few days, varying in different cases, the animals in most cases died. Death was, as a rule, preceded by a short period of dyspnœa, or again the animals may have appeared ill through the whole experimental life. The autopsies revealed in most cases pleurisy single or double, even though injection was made into one side of the chest. The lungs in no case were the seat of complete hepatization (pneumonia) lobar in distribution; but in rabbits 1, 3, 6, 9, and 11 there was a condition of the lung not dissimilar to an engorgement in areas to smaller or greater extent. In rabbit 6 this approached closest the type of hepatization in the upper lobe of the left lung. Pericarditis was present in most and also very marked peritonitis. Care was in all cases exerted to inject the fluid containing the microörganism just beneath the rib into the pleural space, though it was often impossible to say that it had not penetrated the lung; at least care was taken that it should not do so.

In most of the cases the spleen was enlarged to palpably twice or thrice its original size. Cultures made of the blood and pleuritic or pericardial fluids of such animals also yielded a uniformly certain growth of a diplococcus which in every way and reaction corresponded to the diplococcus pneumoniæ of Fränkel and Weichselbaum. Its growth was at first quite vigorous on the Weichselbaum agar, but it died out and was soon lost if not re-inoculated, just as the original pure culture obtained from the empyema pus. Animals injected with the pleuritic or peritoneal fluid of other animals that had died of

injection of pneumococcus also died certainly and even rapidly. The lung and spleen fluids (obtained by expression) with cover-glass stain also showed myriads of these diplococci. By the injection of pure cultures of diplococcus underneath the skin, the results were not so certain, though in some the animals died with the above features. The pus from empyemas known to contain nothing but diplococcus pneumoniæ also killed the animals with unfailing certainty; cultures made from these animals revealed the diplococcus only, and injections of peritoneal fluids from these animals into others proved fatal with results showing pneumonia, pleuritis, pericarditis, and peritonitis, and in all the fluids of such animals, inclusive of the blood, the diplococcus was found. In some cases the animals would seem to have had vitality enough to have resisted this organism, and completely recovered from the effects of their injections and consequent inflammatory disturbances. Such animals were killed after a sufficient period had elapsed, and in most cases there could be seen adhesions of the pulmonary to the costal pleura, and adhesions of the pericardial surfaces. Some guinea-pigs injected gave results much the same as those seen in rabbits. In other cases the animals seemed to have successfully resisted the effects of the micro-organisms, for upon autopsy nothing was revealed that could be traced to any pathological process.

Experiments with the streptococcus which was isolated from the pus of the empyemas recorded in this paper, and which appears to be identical with the streptococcus pyogenes, though positive in some features were yet not so distinctive. The results seemed to vary, for while in some cases streptococci did not prove fatal, in another the effects seemed most virulent and rapidly lethal. In some, injection of streptococci into rabbits had no effect, the animals survived, and being killed after weeks had elapsed, absolutely nothing abnormal was found. In other cases streptococci (from the same empyema) caused marked disturbances; the animal appeared quite ill, but recovered and was for weeks apparently in good health; when killed, a few pleuritic adhesions only were found.

In other cases, an injection of a pure bouillon culture of streptococci, notably in rabbit 15, taken from Case XI., caused the death of the animal in two days, and autopsy revealed nothing but an enormous spleen (the largest I have seen in my experiments) with kidneys palpably swollen; cultures of the blood revealed streptococci. Thus, in this case, we have the symptoms of a pure septic effect, a septicæmia without inflammatory lesions. Again, this same bouillon culture, which was pure in every way and repeatedly tested as to purity by plate methods, was injected at the same time into another animal in the same amounts, though not in the same manner, and there resulted the unusually virulent effects seen in rabbit 16, in which small metastatic abscesses appeared in

different organs of the body, and general jaundice. The spleen also was notably enlarged. The results, though corresponding in certain ways, do not give anything not characteristic of streptococci isolated from other inflammatory processes in the body, and the experiments upon rabbits 15 and 16 agree closely with what is recorded of experiments made with streptococci pyogenes isolated from cases of pyæmia, and notably that of the kind recorded by Baumgarten.

I did not make any experiments with staphylococci isolated in my cases, the reactions of the microörganisms being so familiar in every laboratory. It appeared to me sufficient to establish its biological identity.

The experiments with the pus obtained from the cases of tuberculous empyema were negative. The pus was contaminated at first with streptococcus pyogenes and later with putrefactive microörganisms, and this clouded the reliability of the experiments. Injections into the eyes and pleuræ of animals gave no results that were of scientific value.

Experiments upon Animals.

Rabbits, diplococcus pneumoniæ.

1. Medium-sized, white rabbit, injected in pleural cavity, with a pure culture in bouillon of the diplococcus pneumoniæ; died in eighteen hours.

Autopsy.—Rigor mortis marked; chest on both sides showed pleurisy of a sero-fibrinous character; fibrin of a greenish-yellow tint; serum more abundant in left chest. Pericardium: pericarditis, with slight amount of fibrin. Peritoneum: a slightly increased serum; no fibrin; no evidence of inflammation; Blood gave upon culture and stain diplococcus pneumoniæ (pure). Pleuritic fluid, diplococcus pneumoniæ pure culture.

2. Black rabbit (small). Inoculated March 17th; killed by medulla while dying, March 21st. Injected pure culture diplococcus pneumoniæ in chest.

Autopsy.—Sero-fibrinous pleurisy, double sero-fibrinous pericarditis. Peritonitis, sero-fibrinous exudate. Spleen large and swollen. Blood and pericardial fluid pure culture diplococcus pneumoniæ.

3 .Small white rabbit; injected about ten minims bouillon culture in chest of diplococcus, March 18th.

Autopsy, March 22.—Beginning rigor mortis. Slight pleuritis; very little serum and fibrin in pleuritic cavity. Sero-fibrinous exudate in peritoneal cavity (peritonitis). Spleen not very large. Blood gave pure culture diplococcus in agar and bouillon; no growth in gelatin and found also by stain. Lung juice crude cover-glass gave exquisite capsule diplococci.

4. Rabbit, medium sized, injected with pure bouillon culture diplococcus pneumoniæ; died instantly; failure of injection.

5. Rabbit, medium sized; injected in chest same as 4; died on the fourth day.

Autopsy.—Pleuritis double sero-fibrinous; very severe. Pericarditis sero-fibrinous, pericarditis marked. Spleen large and soft. No peritonitis; no meningitis. Specimens of blood, pleuritic and pericardial fluid inoculated on agar, gelatin, bouillon, etc.; reaction by stain and culture of diplococccus pneumoniæ.

6. Rabbit, medium size, injected in chest with pure culture of diplococcus pneumoniæ; died in six days.

Autopsy.—Double sero-fibrinous pleurisy. Pericarditis sero-fibrinous. Peritonitis sero-fibrinous. Spleen large. Cultures made from blood and pericardial fluid; diplococcus pneumoniæ.

7. Rabbit, medium size, injected subcutaneously with pure culture of diplococcus pneumoniæ; no result.

8. Rabbit, black and white spotted, medium size; injected in chest with five minims of empyema pus in which diplococci pneumoniæ existed; animal died in thirty-six hours.

Autopsy.—Forty-eight hours after injection. Rigor mortis marked. Pleurisy slight; some fibrin in the left side at point where needle entered the lung. Peritonitis very marked with sero-fibrinous exudate; brownish serum in bottom peritoneal cavity. Cultures from peritoneum and pleura, pure culture diplococcus pneumoniæ.

9. Small, active rabbit, injected with pure culture diplococcus pneumoniæ obtained from animal 8.

Autopsy made before rigor mortis. Pleurisy marked; sero-fibrinous exudate on both sides. Pericarditis with adhesions; sero-fibrinous exudate. Spleen large. Peritonitis marked; sero-fibrinous exudate. Pure cultures of diplococcus pneumoniæ obtained from pleura, pericardium and blood.

10. Rabbit, medium-sized animal, injected with peritoneal fluid of animal 9; died in two days.

Autopsy.—Four hours post mortem. Pleurisy left sero-fibrinous. Pericarditis slight. Peritonitis marked. Spleen enlarged. Cultures show diplococcus pneumoniæ.

11. Rabbit, large white doe, injected with twenty minims of peritoneal fluid of rabbit 10, in each lung ten minims; killed by medulla on seventh day in dying condition.

Autopsy.—Marked sero-fibrinous pleurisy on both sides and also pericarditis. Spleen large.

12. Rabbit, small-sized, lively animal; injected with peritoneal fluid of rabbit 8; first two days considerable dyspnœa, but recovered and was apparently well. Killed after a week; the only thing found were a few delicate adhesions between the coils of intestine; some clear fluid in bottom of peritoneal cavity.

13. Rabbit, medium-sized; pure culture injection; killed after fifth day.

Autopsy showed marked sero-fibrinous pleurisy; marked sero-fibrinous pericarditis, also advanced peritonitis, with sero-fibrinous exudate. Large spleen. Pure culture of diplococci obtained from blood, pleuritic and pericardial fluid.

14. Rabbit; died with a paralysis of posterior extremities; exhaustion; remains of an old pleurisy found; adhesions of the costal and pulmonary pleuræ. This animal was injected in the pleura with pure culture streptococcus pyogenes.

15. Rabbit; injected with pure culture streptococcus pyogenes in pleural cavity from Case XI.; died after two days.

Autopsy.—Kidneys larger than normal and congested, but the spleen was enormous in size. Cultures of blood gave streptococci.

16. Rabbit; subcutaneous injection streptococcus pyogenes (pure); death after three days.

Autopsy.—Jaundice, universal, of all tissues. Lungs contained small abscess in lower right lobe. Heart, normal. Liver, very small abscesses, studded throughout. Kidneys, cloudy swelling. Spleen much enlarged. Urine, bile pigment.

17. Rabbit; injected with pure culture streptococcus pyogenes, with no result; lived for months.

In addition to the above experiments, there were some injections into guinea-pigs of the diplococcus pneumoniæ; these resulted negatively, except one animal which had been injected with pleuritic fluid from a rabbit that had died in its turn from an injection of diplococcus. In this there were typical pleurisy, pericarditis, and peritonitis, with large spleen.

A few injections upon rats and mice subcutaneously resulted in case of diplococcus positively in one case. Injections into additional rabbits with pus obtained from a tubercular case gave nothing worthy of note.

Cases.

Case I.—Y. S., aged seven years, admitted into hospital. Mother has had phthisis; child has had measles, four years ago, since then a slight cough. Became sick suddenly two weeks ago; has been ill since with cough, pain in the side, chilly sensations, dyspnœa.

Physical signs (left side). Flatness from mid-scapula down. Bronchial voice and breathing (at middle scapula), below distant breathing. Signs of bronchitis on both sides; no signs of phthisis in front or on opposite side.

Operation eighth rib, resected in the post-axillary line; abscess was found to be higher up and encapsulated; adhesions had to be broken down in order to reach empyema. Examination of pus: crude cover and cultivation; streptococcus pyogenes. Cured.

Case II.—R. T., male, aged eight years; January 1st. Family history negative. He had scarlet fever four years ago; a year ago had a bronchitis; a month ago also bronchitis (?). Two weeks ago had fever, temperature 104° (physician). Complained of headache; next day had pain in the left side; dyspnœa; slight cough; no expectoration; temperature 103°. During the next week the temperature did not exceed 101.5°.

Admission to hospital showed marked œdema of skin of left side, but on incision later (at operation) no infiltration of pus was seen; signs of fluid in left side.

January 2. Operation, resection as noted; there was no infiltration of the œdematous skin with pus. In spite of resection the lung would not expand.

January 3 *to February* 10. Temperature 100°, 101°, to 104°; pulse 110, 112, 138, to 152; respiration 32, 44, to 52. The boy did badly; there was a profuse discharge of pus, fœtid in odor at each dressing, and lung did not expand. Several resections were performed upon this chest.

to favor retraction of chest toward lung and closure of the large cavity in the chest. There resulted much deformity, and at discharge from hospital there existed a discharging fistula in the chest.

Bacterioscopic results were limited to stainings of cover-glass specimens of pus and showed tubercle bacilli. Animal experiments negative.

CASE III.—I. S., aged two years; male; February 18, 1890. Has had measles, a year ago; inflammation (?) of the lungs ten months ago; present illness began with fever six weeks ago; this fever after a few days was complicated by convulsions recurring at intervals. Cough was very marked and present during the entire illness; there was loss of flesh and strength, and increasing palor.

Admission to hospital showed a weakly child, badly nourished, pale; temperature 100°; respirations 40; pulse 120. Physical signs of fluid in right side chest. Tenth rib exsected; about a pint of thick, green, tenacious pus evacuated.

Bacterioscopic result of examination, pus; diplococcus pneumoniæ (Fränkel and Weichselbaum).

May 11. Discharged cured.

CASE IV.—I. S., male, aged eleven months; has been sick for three weeks with fever, cough, dyspnœa, emaciation. Signs of fluid in right pleural cavity. Dulness to flatness. Loss of movement. Loss of fremitus. Loss of voice; pleuritic râles over whole side; syringeful yellow, thick pus removed.

Bacterioscopic examination. Staphylococcus pyogenes aureus. Result unknown.

CASE V.—P. M., male, aged two and a half years; March 3, 1890. Previous history negative. Present illness began a month ago with fever, cough, difficulty in breathing, and constitutional disturbances; fever continued marked during the first two weeks, then abated, but continued nevertheless; palor, loss of flesh and strength, and generally becoming worse.

Admission to hospital. Anæmic, poorly nourished, anorexia, cough, prostration, and fever; temperature 101°; respirations 46; pulse 140. Signs of fluid in the left side and areas of consolidation (broncho-pneumonia) over the right side. Heart apex just inside the middle line.

March 4. Turbid flocculent serum removed from the chest. Cultures show diplococcus pneumoniæ. Aspiration, only four fluidounces (serous) removed in all, but patient continued to grow worse, temperature varying from 101° to 104°; respirations from 50 to 70; pulse 148 to 160.

16*th*. Needle in left side withdrew a creamy pus.

18*th*. Resection of rib, and about one ounce of pus evacuated, but patient grew worse; died March 27th from broncho-pneumonia on both sides.

Examination (bacterioscopic) of pus withdrawn March 16th, showed diplococcus pneumoniæ.

CASE VI.—I. K., male, aged three and a half years; March 13, 1890. Family and personal history not lucid. Present illness began nine weeks ago with fever and cough and difficulty in breathing; prostration, loss of appetite; patient complained of pain in the left side; loss of flesh and strength; there were night-sweats; patient has steadily lost ground.

Admission to hospital. Poorly nourished, anæmic, delicately built lad; no sign of rhachitis; temperature 101°; respirations 40; pulse 132 (dyspnœa). Physical signs showed fluid in left side. Heart apex

pushed to the right, and most intense behind lower end of sternum; no signs of phthisis at apices.

March 16. Operated resection of eighth rib; greenish odorless pus, with large masses of fibrin evacuated, as big as a child's hand.

28*th*. Discharged cured.

Bacterioscopic examination of pus, diplococcus pneumoniæ.

CASE VII.—M. B., aged two years; male; March 1, 1890. Family and personal history negative. The previous history is indefinite; the patient for some time has suffered from cough. Present illness dates a week back; contracted a severe cough, and dyspnœa became very marked; fever, night-sweats, loss of appetite.

On admission, patient is well nourished; suffers great dyspnœa, this symptom so marked as to give impression that œdema of the glottis was present, or obstruction in the larynx. Intubation attempted but failed, and tracheotomy performed. Temperature 100°. Examination of the chest revealed spots of broncho-pneumonia over both sides. March 5th to 15th, temperature 101° to 105°; respiration 40 to 76; pulse 140, with dyspnœa; increasing; attempts to remove tracheal tube resulted in renewed attacks of dyspnœa. March 15th, signs of fluid in left side of chest, and on March 16th, operated, and twelve ounces of pus, greenish, no bad odor, removed from left side. Eighth rib resected.

March 18. Discharged cured.

Results: Bacterioscopic study and culture of pus. Diplococcus pneumoniæ.

CASE VIII.—H. G., male, aged twelve years. Father, mother, and sisters and brothers in good health; previous history indefinite. It is not possible to obtain definite information as to the exact time of onset of present illness. He has for a few weeks past suffered from cough, fever, and night-sweats, pain in the right side upon coughing or taking deep inspiration; has been confined to his bed; no history of traumatism.

Admission to hospital. An anæmic but well-built lad, not at all emaciated. Physical examination reveals fluid, and needle pus in right side. Temperature 100°; respiration 40; pulse 140; resection of rib on right side and a pint of greenish fluid, not adhesive pus, removed; no fibrin clots were withdrawn; some adhesions between the costal and pulmonary pleuræ.

Bacterioscopic examination of pus revealed streptococcus pyogenes.

Result: Cure.

CASE IX.—Female, aged two and one-fourth years. April 10, 1890. Family history shows nothing. Present illness began three weeks ago with some eruption (?) upon the body; about a week later there appeared a cough, fever, distress in stomach and abdomen.

Admission to hospital. Anæmic child, with signs of rhachitis, cough dyspnœa, and some cyanosis; temperature 101°; pulse 160; repirations 48. Physical signs of fluid in the right side, pus withdrawn, immediate operation, exsection; sixteen ounces of pus evacuated. April 12th to 22d, temperature varied from 99° to 103°; respirations 40 to 43; pulse 130 to 158; but April 22d, symptoms pointing to an invasion of the healthy lung by broncho-pneumonia appeared, and patient died with all the symptoms of pneumonia and exhaustion.

Bacterioscopic examination of pus revealed diplococcus pneumoniæ (Fränkel and Weichselbaum).

CASE X.—M. G., male, aged four years; April 18th. Family and

previous history negative. Present illness began seven weeks ago with scarlet fever; three weeks later the patient developed general anasarca, this lasted until eight days ago; at the same time he had a severe cough, chills, and fever; constant sweating, lasting until the date of admission; loss of flesh and strength, also failure of appetite and frequent micturition.

Admission to hospital. Patient anæmic and emaciated, cough and dyspnœa. Temperature 99°; respirations 42, and pulse 140. Signs of fluid in the left side. Heart apex pushed to the right of the sternum.

Operation, exsection of rib; twenty-nine and a half ounces of pus of creamy character evacuated.

Bacterioscopic examination revealed diplococcus pneumoniæ.

Result: recovery.

CASE XI.—Male child, aged four months. May 9, 1890. Had been perfectly well until two weeks ago, at this time child was vaccinated; simultaneously with the vaccination, there appeared a burrowing abscess on the dorsum of the left foot which was deep and passed between the metatarsal bones. Eight days ago a febrile movement appeared and the patient was referred by me to a surgeon for treatment. There were at this time no lung symptoms. Three days ago the child developed cough and dyspnœa.

Status: There is cough, dyspnœa, sighing respiration; pulse increased in rapidity; temperature 103°; abscess on foot still present; vaccine pustule still size of five cent piece and angry-looking. Examination of left side revealed chest full of fluid and needle withdrew pus, light yellow, thin, easily separating into two layers, clear and serous, upper and lower purulent.

May 10. Exsection of rib; six ounces of pus (milky) evacuated.

12*th*. Died; no autopsy.

Bacterioscopic examination of pus revealed streptococcus pyogenes.

CASE XII.—*May*, 1890. Male, aged twelve months; well developed; has been suffering from pertussis and just recovered; about two weeks before date was doing well, when suddenly there appeared a new cough with dyspnœa, and broncho-pneumonia of the right lung was diagnosticated. The cough and fever and dyspnœa still continued up to May 9th, when fluid was found in the right side. A needle introduced about this time withdrew a clear serum; temperature 103°; pulse 160, and rapid respiration. Transferred to the hospital, where child next day showed a measles eruption.

13*th*. Needle introduced into right side and pus was withdrawn; child was doing well, but on the morning of the above date, patient suddenly developed tympanites (peritonitis (?)) and died.

Bacterioscopic examination of pus of May 13th and serum of May 9th revealed diplococcus pneumoniæ.

CASE XIII.—Empyema; perforation; spontaneous recovery. Female, aged thirteen months. May 12, 1890. Previous history negative. Child was taken ill about three weeks ago with cough, fever, dyspnœa. The child had dyspnœa, and moaned when it breathed; there was a frequent cough and a temperature of 103½°; pulse 160. Examination of chest showed consolidation of the left upper lobe (dulness, bronchial voice and breathing). Diagnosis: broncho-pnumonia. The patient seemed to improve up to a week ago, when the temperature and cough persisted, the dulness behind became more marked and spread toward the base

of the lung; fremitus absent; voice and breathing bronchial. Needle introduced and pus withdrawn from left side, greenish-yellow, adhesive; heart not displaced; operation advised.

August 1. Child had escaped from observation, and having refused operation passed under care of others. It was brought to me, August 1st, for diarrhœal trouble; chest showed no signs of old trouble; lung had expanded; voice and breathing good, no friction sounds. There was a slight dulness only left over the affected side; fremitus good; no fever; child looks much improved. Mother says that after leaving my care the patient grew worse until she began to cough up large amounts of yellow matter (pus?). This continued some time at intervals; patient recovered; no cough.

Bacterioscopic examination of pus May 12th: diplococcus pneumoniæ.

Case XIV.—*May* 13, 1890. L. H., aged eighteen months, male; previous history negative; father and mother well and in apparent good health. Nine days ago child developed a cough, fever and dyspnœa; the dyspnœa increased, fever continued and there was restlessness at night. When brought to me the child was anæmic; had signs of rhachitis; there was much dyspnœa and moaning respiration, drowsiness, and partial stupor. Temperature 105°; pulse 165; respirations 56. Physical signs of fluid in right side below mid-scapula, needle introduced and removed, a clear bloody serum obtained.

14*th*. Needle introduced, signs of fluid having increased, and withdrew milky pus.

15*th*. Exsection of rib, eight ounces of pus removed.

Bacterioscopic examination showed staphylococcus pyogenes aureus.

Case XV.—D. H., male, aged five and a half years. Father and mother living, has had no special illness; present illness dates from October 6th, when there was fever and pain and dyspnœa; the pain was referred to the stomach; there was no distinct history of chill. When first seen, October 7th, patient was a well-developed child; no signs of past rhachitis; there was dyspnœa, a dry cough, fever; there was also some restlessness; complains of stomach pain. Temperature 103°; pulse 130; respirations increased. Signs in chest negative.

October 8. Temperature 104°; pulse 140; respirations rapid; pain in right side; rusty sputum. Diagnosis at this time; right lobar pneumonia lower lobe. At end of five days signs pointed to a resolution of an ordinary lobar pneumonia; temperature 102°.

19*th*. Signs of fluid in chest. The boy has not been doing well, and new signs appeared; loss of fremitus; flatness, bulging of right side; subcrepitant râles close to the ear, voice and breathing though coarse heard over whole chest; dyspnœa great; temperature 103½°; pulse 160; respirations rapid.

21*st*. Operation: simple opening in chest and tube inserted; about half a pint of pus evacuated; not fœtid; yellow color.

27*th*. Discharge fœtid and penetrating odor; the drainage bad and a resection was performed, November 5th. Final complete recovery.

Bacterioscopic examination of pus first taken from chest showed diplococcus pneumoniæ.

Clinical Classification.

The cases here recorded, from a clinical and bacteriological standpoint, divide themselves quite readily into groups.

Group I.—Here we can place those cases of empyema in which the bacterioscopic examination revealed the staphylococcus pyogenes aureus, or the streptococcus pyogenes. These microorganisms did not exist associated, but in so-called pure form and isolated in the exudate. The etiology of these cases is very difficult to make out, because this group does not include cases of empyema where an extraneous source of infection exists. Those cases of empyema in which the pleuritic exudate shows simply the presence of the staphylococcus or streptococcus alone, and where there is no history of traumatism with perforation of the skin, or where there is no suppurating focus of infection outside the pleural cavity, are most puzzling to explain because the bacterioscopic finding gives us no clue as to the origin of the disease. The staphylococcus and streptococci are microörganisms which exist in suppurating processes of the most diverse nature and situation in the body. I have four such cases to place under this heading. One female, aged seven years, and three male children, aged eighteen months, two years and twelve years respectively. In none of these cases was there a history of traumatism of any kind. When they came under observation, they had been ill with symptoms of pulmonary trouble for periods varying from one to six weeks.

Assuming that an infection of the pleura is most apt to result from adjacent inflammatory processes, the most usual disease preceding or complicating such a pleurisy is pneumonia. Weichselbaum has demonstrated that in addition to the pneumococcus of Fränkel, there exists in the lungs of pneumonic patients the streptococcus pneumoniæ (or pyogenes) and the staphylococcus pyogenes aureus. Though we cannot as yet yield to these microörganisms the dignified position of being the direct cause of pneumonia, we find that they exist in the lung constantly as so-called mixed infections, as they do in diphtheria. Thu[1] has argued that microörganisms in pneumonic fibrinous pleuritis and pericarditis (pneumococci) first find their way through the lymph channels into the subpleural tissue and then into pulmonary pleura, and finally gain the surface of the pulmonary and costal pleura; from thence they may be carried into the mediastinal spaces and reach the tissue of the pericardium and even the muscular tissue overlying the costal pleura. It is true that the failure to find the pneumococcus in the exudate does not disprove the possibility that at some early stage of the empyema it might have been present; however the tendency to-day is to consider the active

[1] Centralbl. f. bakteriol., 1889, Bd. v.

predisposing causes exposure to cold and moisture. It is thought that though the above microörganisms may exist in the air passages, as has been proven in the nose and mouth of a healthy individual, they may remain inert until, cold and wet reducing the resistant vitality of the organism, they cause an empyema; so a wound in the chest wall without perforation of the skin may exert a similar influence.

GROUP II.—The cases of empyema which fall under this group are the most interesting of all pleurisies. They are those which either complicate or follow a pneumonia. In the pleuritic exudate of such an empyema the diplococcus pneumoniæ (Fränkel and Weichselbaum) may be demonstrated as in all of my cases.

These were two female children, aged thirteen months, and two and a quarter years, respectively, and seven male children aged twelve months, two years, two two and a half years, three and a half years, four years, and five and a half years. They had been ill when they came under observation for periods ranging from one to nine weeks. In one case the patient had been under the observation, from the very beginning, of a skilful observer (Case XV.), and the diagnosis of lobar pneumonia followed by a pleurisy (empyema) was distinctly traced. As recorded elsewhere, the diplococcus pneumoniæ (Fränkel) could be easily recognized in the crude pus by spreading upon cover-glass; beautiful capsule appearances were also obtained. Pure cultures were isolated in every case and their virulence tested upon animals while the cultures were still recent. The experiments were in the direction of intra-pleural or pulmonary injections, so that appearances in the animal experiments resembled very much what is seen in the human subject. If such a pathogenic microörganism is found in a pleuritic exudate, it can be traced to only one probable source, the lung. It is scarcely necessary in any of these cases to prove clinically the existence of a pneumonia. If one will refer to the histories of the cases he will see that in some a pneumonia, either of the lobar or lobular type, must have been in active progress when the empyema was operated upon. At least the physical signs and symptoms indicated this condition of affairs in the lungs upon the opposite side to the empyema, if not in the lung corresponding to the empyema.

In one case, where the patient succumbed, the opposite lung was undoubtedly affected. In other cases, after operation, the temperature would not only persist, but persistent broncho-pneumonic processes were present; this was notably the history of Case III., who continued in the hospital for months, being finally discharged cured. In these cases, tuberculosis was excluded by all possible methods, both clinical and bacteriological. But in the majority of the cases in which the pneumococcus was found in the pus of the empyema, the evacuation of the pus

marked the retrograde of all symptoms and recovery of the patient, showing that the pneumonia, if it existed, had preceded the accumulation in the chest and had passed through its various stages. The pneumococcus has been found by Weichselbaum in lungs the seat of lobular pneumonia, as well as in those the seat of lobar process.

I have elsewhere described the macroscopic characters of the pleural exudates in the cases of empyema belonging to this group. But in two the pleuritic fluid when first withdrawn from the chest was serous in character, in one case even devoid of flocculi. In both of these cases, the exudate taken later with an exploring syringe was markedly purulent, and both cases were operated upon. In the serous fluids first obtained, as also in the purulent exudate, the diplococcus was found. This shows distinctly that though an exudate may be serous at first, it may subsequently become markedly purulent, not on account of anything introduced into the chest on the occasion of the first puncture, but from the continued action of microörganisms already present (diplococcus pnemoniæ or streptococcus pyogenes, Fränkel). Again, pus in the chest has a tendency, without this cavity, to separate into strata, the upper one being serous and containing but few leucocytes; we may aspirate this stratum in a marked empyema, and thus can not draw any definite conclusion as to the nature of the exudate. If an exudate contain streptococci, even though serous, we can with certainty predict the advent of pus (Fränkel). We might make a similar assertion of fluids containing the diplococcus pneumoniæ. If a serous fluid withdrawn from the chest fails to reveal any microörganisms upon stain or culture, we can conclude that there is a probable tubercular element in the pleurisy (Fränkel). If such a serous exudate subsequently shows the presence of microörganisms (streptococci) and then becomes purulent, we can suspect contamination. With conscientious cleansing of a needle-syringe, we may fearlessly enter the pleural cavity without having in the least compromised the health of the patient. I am certain much misunderstanding has arisen on this subject through a misinterpretation of the real nature of some of these serous exudates. While some are devoid of microörganisms (tubercular), others are filled with pyogenic microbes, capable not only of producing suppuration, but maintaining it.

GROUP III.—This group, which includes empyema of tubercular nature, is most unsatisfactory in its various aspects. Fränkel, who spent much time and patience upon tubercular pleurisies in the adult, found cases in which examination of the exudate yielded a negative result. So common was this, that he concluded that a negative finding was much in favor of the tubercular character of the exudate. These exudates may be localized or involve the whole pleura. They may or may not be accompanied by lung areas of tuberculosis; in cases where the lung is involved, in the adult, the tubercle bacilli may be found in the sputum,

but in children the examination of the sputum is not always practicable. If, as in children, the lung shows no positive involvement and the examination of the exudate is negative, diagnosis and prognosis are difficult. Ehrlich (*Charité Annals*, 1888) explains the absence of tubercle bacilli in two ways: 1. The fibrin formations in the exudate remove the bacilli by enclosing them. 2. Thickening of the pleura by adhesions causes a resistance to the transmigration of bacilli. In empyema, the bacilli are more numerous than in simple pleuritic exudates, because cells passing in myriads into the exudate are more apt to carry bacilli with them. Israel and Gerhart think that the bacilli rather become entrapped in the miliary growths of the pleura, and thus do not pass into the exudate.

I have but one case to place in this group, that of Case II., a boy of eight years, who had had several bronchitic attacks years before the advent of his empyema. The exudate had to be repeatedly examined in order to establish the presence of tubercle bacilli (stained by Ehrlich's method), but the experiments with animals were of a negative character. In this case the streptococcus pyogenes was also found in the exudate from the very first; this is similar to a case in the adult recorded by Fränkel. The formation of adhesions tying down the lung must have been exceedingly great, for operation found the lung unable to expand and repeated exsection of the rib had to be resorted to, in order to obtain partial closure of an immense suppurating cavity. The deformity resulting was most pronounced. I have made attempts in other children and this boy to obtain sputa for examination, but have not succeeded. I attributed my ill success in inoculation upon animals to the presence of contamination in the pus.

Thus, my group of tubercular empyema remains unsatisfactory, though diagnosticated with a certainty from the presence of tubercle bacilli in the exudate. The lungs upon examination yielded no evidences of involvement.

Group IV.—In this group we might class those cases of empyema in which some focus of suppuration situated in another part of the body may be with great probability pointed to as a source of infection. The infecting focus may be adjacent, as in the cases of Fränkel, where a retro-pharyngeal abscess or perforating peritonitis were the cause of the empyema, or the empyema may be one of the manifestations of a species of pyæmia (as in my own case). This was a male infant, aged four months, who was perfectly well up to within two weeks of its death. The infant was vaccinated and then developed a burrowing abscess of the foot, and after this the empyema appeared. No autopsy was allowed; it would have been interesting to see (though no pneumococcus was found in the exudate of the pleura) whether a pneumonia (pyæmic) was present with the empyema. The rapid death of the

child speaks in favor of some virulent infection; an autopsy might have revealed several other hidden foci of suppuration.

The bacterioscopic finding and experiments in the above case are interesting. The streptococcus which was isolated and existed alone in the exudate was certainly very virulent. Two animals injected with a pure culture (15 and 16), both died. Both were injected at the same time with the same culture; the one in the pleural cavity, in this case, the animal died in two days; streptococci were found in the blood and organs, but there were no inflammatory exudates; the kidneys were swollen and the spleen was enormous in size. The second animal was injected subcutaneously, and the report of autopsy shows metastatic abscesses all over the body with general jaundice and enlarged spleen and cloudy kidneys. These results differ widely from those with streptococci obtained from cases of Group I. Here the animals survived and autopsies after months showed no effects of infection, or as in rabbit 14, only the effects of local processes limited in nature.

Putrid Empyema.

By this, I desire to designate those cases of empyema in which there was a putrid or fœtid odor to the discharge, and in which this odor persisted.

In both of my cases the exudate turned putrid after operation. In one (XV.) the empyema followed a pneumonia, and in the fluid first withdrawn from the chest, the pneumococcus alone existed. In this case, on account of the imperfection of drainage due to the first mode of operating, there was retention of pus, the chest wound finally closed, and when reopened again for resection, the pus was very putrid.

The second case was that in which the pus showed the presence of tubercle bacilli. There was no odor to the pus upon the first resection. But there was not only lack of any expansion in the lung, leaving an immense cavity in the chest, but the pus remained in this cavity. Here the emyema became putrid, necessitating repeated washings of the chest with creolin solution. In spite of this fact, the pus remained putrid. Investigation succeeded in revealing the presence of a florescent green bacillus, rather thick and short, which fluidified gelatin (much like that found in water) and grew upon agar of Weichselbaum in a dark-green layer with a rather mawkish odor to the growth. This is all that could be found in addition to the tubercle bacillus and streptococcus previously present. This shows that the most innocent exudate may turn putrid, as in the case of pneumonic empyema, as well as the tubercular cases. It seems rational to suppose that lack of expansion of the lung or retention of pus, especially where the chest has been injected with so-called antiseptic solutions (in the above cases boric acid or creolin), are conditions peculiarly

favorable to the development of putrid exudates. I have seen air enter daily freely into the chest cavity without causing the pus to become putrid; also the introduction of bacteria from the lips of the wound was unavoidable, on account of the free moving of drainage-tubes and dressings and tampons over the mouth of the external wound. Yet drainage being free, with full lung expansion, no putridity appeared. In the future, the appearance of a putrid exudate in a chest in which primarily the pus possessed no such characters, would make me think of retention due either to insufficient drainage or want of proper expansion of the lung.

PROGNOSIS.—We can safely say that the present status of our knowledge in these cases enables us to assure our patients with moderate certainty as to the outcome of the illness of their children. Such prognosis may be made at the bedside or within a very short period from the first examination of a case. The simple means at the disposal of every clinician are adequate for the examination of crude specimens of pus, and delay of a day or two at the most is all that may be necessary for more deliberate investigation. Nor do I think that we should take too much credit to ourselves for the recovery of a certain class of these cases which, under proper guidance, invariably get well. Thus, it would appear that the best prognosis is held out to those which belong to Groups I. and II. More especially is this true of cases of meta-pneumonic empyema. In one of these (XIII.), in which the purulent exudate filled the chest and was withdrawn from the same by exploring needle and contained pneumococci, the patient recovered; having refused operation, the empyema perforated the lung and thus gained exit externally, pus being coughed up. Subsequent examination of this case revealed nothing abnormal on the diseased side but slight dulness. The lung had thoroughly expanded. Fränkel records similar cases in the adult. It would scarcely be well to expect too much from such "laissez faire" treatment, for a large percentage (Ziemssen's *Vorträge*) of cases which are allowed to perforate die. The age of the patient and the presence or absence of any considerable pneumonia will weigh in each case. Some authors are enthusiasts for the simple opening and drain in the chest; others insist on the trial of repeated aspiration; others contend that resection of the rib is the only safety of the patient and secures a rapid expansion of the lung. The truth lies in the etiology of the cases of empyema. In the meta-pneumonic exudates cases will do equally well by the various modes of treatment; in other words, the pus evacuated by whatever means, there is an inherent tendency to absorption of what inflammatory product there is remaining and return to a normal status, as in the pneumonic lung.

The pneumonic exudates, if they hold out such brilliant prospect of recovery to the patient, are a striking contrast to the tubercular cases,

where only a partial recovery is at best possible. There are cases on record where, especially in children, an empyema (Ziemssen's *Vorträge*) of established tubercular nature has made an apparent good recovery, yet we must doubt its permanency. We cannot rid ourselves of the idea that a large extent of surface like the pleura, once tubercular, must, if recovery takes place, be a latent danger to the patient.[1] In other cases, the recovery results with fistulæ, retraction and all the concomitants of permanently crippled respiratory apparatus. The pyæmic cases are fatal, as far as we know.[2]

[1] Klesch and Vaillard (Arch. de Physiol. et Path., 1886) have made autopsies upon pleurisies of a tubercular character which had apparently recovered, and were able to prove their tubercular nature at autopsy.

[2] I desire to express my sincere obligations to Dr. Barium Scharlau, of the Mount Sinai Hospital, for the clinical material which he so kindly placed at my disposal.

ON THE

ETIOLOGY OF DIPHTHERIA.

AN EXPERIMENTAL STUDY.

BY

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ETIOLOGY OF DIPHTHERIA.

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The thoughtful student of diphtheria is early impressed, alike in his studies by the bedside, at the dissecting-table, and in the laboratory, by the indefinite conception which so widely prevails as to the nature and limitations of the disease. The difficulties with which this lack of precision invests the subject are especially felt when he is trying to get light upon its etiology. The standpoint from which these studies are undertaken can, perhaps, be best made clear by a brief statement of the views which the writer believes to be generally held in this country as to the general nature of diphtheria and its relations to diseases not infrequently confounded with it.

Diphtheria, in its more pronounced forms, is an acute infectious disease, presenting in most cases a more or less characteristic local lesion in some of the external or internal surfaces of the body. This local lesion is most frequent in the pharynx and upper air-passages, but may occur on any mucous membrane, or on the skin, or on the surfaces of wounds.

This local inflammation may find expression in a variety of ways. There may be a simple redness of the affected surfaces, which passes away leaving no trace. There may be a more or less intense catarrhal inflammation with much or little exudation, and nothing else. There may be a limited or extended superficial or deep necrosis of the mucous membrane, a form of necrosis called coagulation-necrosis, in which the nuclei of the cells disappear, and the cell bodies are converted into a more or less homogeneous or granular mass somewhat resembling fibrin, so that the affected surfaces appear to be covered with a whitish or grayish pellicle or membrane. There may be, in connection with or without

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this necrotic pellicle, a fibrinous exudate intermingled with leucocytes, fragments of epithelial cells, red-blood cells, and granular matter.

The pellicle, or false membrane, pseudo-membrane as it is called, may be loosely attached to the affected surfaces, or so firmly adherent as to seem to form a part of the underlying tissues. Associated with all these forms of the local lesion of diphtheria are varying, but usually large, numbers of dead and living microörganisms. Disintegration and softening or exfoliation of the false membrane, with or without loss of substance of the underlying tissue, ulceration, phlegmon, œdema, and abscess, represent occasional progressive phases or complications of the lesion. The adjacent lymph-glands are apt to be swollen and congested.

It is not necessary to describe here in more detail the minute structure of the false membrane in diphtheria, nor the various minor modifications which it presents. Nor is it necessary to do more than call attention to the fact that, although the occurrence of a false membrane is very frequent in the disease and of great diagnostic importance, there are cases of genuine diphtheria in which no false membrane is formed.

It is, however, highly important to remember that, so far as its anatomical characters are concerned, a false membrane very similar to that above described is by no means limited in its occurrence to diphtheria. A pseudo-membrane may be induced experimentally in animals, as it would seem, by any agency which will deprive mucous membranes, such as that of the trachea, of their epithelial covering, and fulfil the conditions of fibrin-generation through the induction of emigration of leucocytes. It may be formed under the influence of a great variety of local irritants, such as steam, cantharides, corrosive sublimate, arsenic, sulphuric acid, ammonia, etc. It may also be formed as Huebner (1) has shown, by such a local temporary mechanical interference with the circulation in the affected part as will impair the integrity of its vascular endothelium.

Under all of these conditions, then, a false membrane may be formed essentially similar in anatomical character, so far as we yet know, save, perhaps, for the microörganisms which it contains, to some of the forms which are frequent in diphtheria. This false membrane is often called a croupous membrane, and the form of inflammation which it accompanies a croupous inflammation. It is also, unfortunately, termed a diphtheritic membrane alike whether it occurs in diphtheria or in inflammation due to a variety of other local causes.

The attempt to establish an anatomical distinction between those forms, especially of pharyngeal and laryngeal inflammation with the formation of a false membrane, which, on the one hand, seemed to be of a purely local character, and, on the other, were associated with evidences of systemic infection; the attempt, in other words, to establish a morphological criterion for the differentiation of what is known as croup or

simple pseudo-membranous inflammation of the larynx and of the pharynx from a more serious form of disease called diphtheria, was a legitimate attempt to meet an urgent demand for precision in the interests both of treatment and prophylaxis. Especially under Virchow's teaching, it was at one time generally believed that, if the pseudo-membrane had evidently encroached upon the structure of the underlying parts, so that its removal involved a loss of substance, its formation was due to those agencies which induced the disease known as diphtheria. If, on the other hand, the pellicle was superficial, it was the result of a simple local croupous inflammation, and might be due to a variety of causes. When, however, it became evident, as it soon did, that in very many cases in which a more or less extensive pharyngeal or laryngeal inflammation with a pseudo-membrane, was associated with grave systemic reaction, the false membrane was apparently entirely superficial, and could be stripped off without injury to the underlying tissue; when, on the other hand, a similar local inflammation developed with no evidences whatsoever of systemic infection, and with no tendency to the spread of the disease from one individual to another, and still it was found that there might be a close adherence, even a genuine infiltration of the underlying tissue with the false membrane: it became evident that the deep or superficial character of the lesion was a distinction between simple pseudo-membranous pharyngitis or laryngitis and diphtheria which, in fact, did not distinguish.

In the reaction which followed the loss of faith in this anatomical distinction between simple croupous and diphtheritic inflammation, the often repeated observation that cases running the ordinary course of a simple local croupous inflammation with no systemic reaction, might apparently serve as a starting-point of widespread epidemics of genuine diphtheria, led many to take the ground that every inflammation with the formation of a pseudo-membrane which was not evidently traumatic —*i. e.*, due either to chemical or mechanical injury—should be regarded as diphtheria. In other words, the view seems generally to have become prevalent that it is at least safe and wise to consider those forms of disease commonly known as simple croup or pseudo-membranous laryngitis and pseudo-membranous pharyngitis as all local lesions of diphtheria, but diphtheria in which the symptoms of general infection are sometimes well defined and sometimes very little marked or absent altogether.

Whether the material which induces the inflammation in diphtheria sometimes reaches the point of local expression through the blood-vessels, or always from the surface in the affected region, it is not possible now absolutely to say, but both clinical and experimental evidences speak strongly in favor of the latter view.

The observations, largely clinical, but in part anatomical, which have led to the general belief that diphtheria is an acute infectious disease

are too well known to need recapitulation here. If it be an acute infectious disease, the great advances in our knowledge of other diseases of this class would justify us in the conjecture that it is caused by bacteria and by some one or more particular species. Should such species, proved to stand in an etiological relationship to the disease, be ultimately discovered in the local lesions, most of the difficulties of definition which now beset our path would be largely removed. In other words, it is the etiological factor in diphtheria which we need to know in order to make those exact distinctions between it and other diseases with a similar local lesion, which the exigencies both of prophylaxis and treatment demand. When we know what causes diphtheria, then, and not till then, shall we know what diphtheria really is and how to define it. In the meantime we have to search for the etiological factor in the disease, without a more precise characterization of it than is furnished by the clinical observations in each particular case.

A large number of the older observers, in the use of the comparatively crude methods formerly in vogue, found bacteria of various forms in the membrane, and most of these seemed to belong to the group of micrococci, although bacilli are noted. But we have long since learned that the morphological examination does not suffice for the determination of a bacterial species. We must cultivate them in pure form to learn the much more definite biological features which characterize them. Not less indefinite were the results of the early attempts to reproduce the disease in animals. For the most part, the inoculation of animals, either subcutaneously or on the mucous membrane or directly into the blood, was practised with fragments of the false membrane, or with cultures from these, which, owing to the lack of proper methods, were never pure cultures of a single species, but a mixture of various forms. Under these conditions, sometimes in the animal a false membrane, or what passed for such, was produced; sometimes fatal septicæmia or local inflammatory lesions; sometimes all of these together, and sometimes none of them. But we should not forget that neither a false membrane nor septicæmia nor a simple local inflammation, nor even the simultaneous occurrence of these necessarily constitutes diphtheria.

We now know that there are many forms of bacteria which, when introduced into the bodies of animals, produce serious disturbance, marked disease, and even death. We know that some of the bacteria which are frequently present in the healthy human body, or its excretions, may, when introduced into the tissues or blood of animals, induce disease and death. So that now-a-days, when animals are inoculated with such unknown mixtures of various kinds of germs as may be in a diphtheritic membrane and die, our interpretation of the results is much more guarded and our conclusions much less sweeping than was formerly the case.

For our present purpose, then, we may pass without detailed review the long list of patient researches on the etiology of diphtheria which preceded the formulation of the new methods of bacterial study by Koch; not because they are without value or importance, but because the vantage-ground of the modern observer is on a so much higher plane of exactness and precision.[1] It implies no lack of respect for the conscientious workers of an older period of science, that at the advent of a new and revolutionizing mode of research the earlier results should for the moment be set aside until in the light of new and rapidly accumulating facts their significance can be justly estimated.

Historical Summary.

The first exact study of the etiology of diphtheria by the use of the modern culture methods was by Loeffler, in 1883 (2). At this time, after a careful *résumé* of the work which had gone before, this observer came to the conclusion that no reliable data were at hand for fixing upon any particular species of bacteria as the cause of diphtheria; nor had any of the variously modified inoculations of animals, either with the crude membrane of diphtheria or the impure cultures from it, ever induced what could fairly be regarded as real diphtheria.

It will suffice for our purpose to give an outline of Loeffler's results. Two species of bacteria were found by him, sometimes alone, sometimes together, in so many cases as to render them of apparent significance. These were: 1, a chain coccus or streptococcus; and, 2, a slender bacillus.

So far as the chain coccus is concerned, although he apparently found it present by the morphological examination, in a considerable number of cases—as well in the blood and viscera as in the false membrane—he isolated and identified it by cultures in only five cases; from the tonsils in three of them; from the heart, liver, and spleen in two; from the kidney in one. Not only the biological characters of the chain coccus, but its general effects upon inoculated animals would seem to indicate its close relationship, if not its identity, with the streptococcus pyogenes and streptococcus erysipelatos. Its local effects upon inoculated mucous membrane did not at all resemble the characteristic common local lesions of diphtheria. Loeffler is disposed to consider this species, in most cases at least, as of secondary importance—though perhaps inducing complications; but his reasons for this view do not seem to be very precise or convincing.

The bacilli of Loeffler are about as long on the average as the bacillus tuberculosis, and about twice as thick. They are frequently a little

[1] For a *résumé* of older researches on the etiology of diphtheria see Loeffler's studies, Bibliography, No. 2.

thickened or knobbed at the end. They stain readily with alkaline methylene-blue solutions. They are immobile, and do not grow at the ordinary temperature of the room. On blood-serum or on a mixture of blood-serum with beef-tea, peptone, grape-sugar, and salt, they grow readily at 37° C. (98.6° F.). They grow, also, as has since been shown, on nutrient agar, to which ten per cent. of glycerine has been added. These bacilli were obtained in pure culture from the false membranes in six cases of diphtheria. In one of these cases the streptococcus was also found. In four of them the material examined was the membrane simply, taken from children during life. By a simple morphological examination of seven other cases he found bacilli which appeared to be similar to those which he had cultivated. In the cases, however, which were examined both morphologically and by cultures, the bacilli do not seem to have been more frequently present than were the streptococci.

Cultures of the bacilli injected subcutaneously into guinea-pigs killed the animals within two to three days, producing a whitish or hemorrhagic exudate at the seat of inoculation and widespread œdema of the adjacent subcutaneous tissue. The internal organs were free from the inoculated bacilli, which were present only at the seat of inoculation. Introduced into the mucous membranes of rabbits, guinea-pigs, chickens, or pigeons, a fibrinous pellicle was frequently formed over the inoculated surface. Sometimes there were considerable general œdema and pleural exudation. On the other hand, the bacilli were not found in all typical cases of human diphtheria. They were absent in more than one-half of those examined morphologically, and in four out of ten in which cultures were made. The bacilli were not present in the fibrinous pellicles induced on animal mucous membrane either in numbers or situations corresponding to the human local lesion. They were often absent altogether. They did not seem to produce their effects on uninjured mucous membranes. They were found once in the mouth of a healthy child.

Thus while the researches of Loeffler led the way along lines which may be regarded in many respects as models for future workers, no positive conclusion was reached as to the cause of diphtheria. He concludes that while his bacilli are possibly the cause of diphtheria, the strict proof was not furnished by his investigations.

Loeffler is disposed to believe that there may be more than one kind of diphtheria; that the form of the disease in which the streptococci are present is characterized by a marked necrosis of the affected surface, while the pseudo-membrane is not prominently or extensively developed; and, further, that the cocci enter the lymph channels and may find their way into various distant parts of the body, even blocking the smaller bloodvessels. That form of diphtheria, on the other hand, in which he finds the bacilli is characterized by an abundant pseudo-

membrane which may contain the bacilli in that part which borders on the original epithelial surface.

This view, that diphtheria might be caused by different species of microörganisms, had been already urged by Klebs (3), who had observed that the bacteria common in the false membrane in the cases which he had studied in Prague were of an entirely different form from those which he found in his later observations at Zurich. While in the former cases they were micrococci (microsporon diphtheriticum), in the latter they were bacilli apparently identical, morphologically, with those which the studies of Loeffler led him to regard as of much significance. The bacilli are now commonly known as the Klebs-Loeffler diphtheria bacilli.

In 1884, Emmerich (4) announced himself at the International Congress of Hygiene at the Hague as the discoverer of the real germ of diphtheria. He considers the disease in man and the pigeon as identical. His bacillus is short and stout, about twice as long as broad, is apt to occur in pairs. It grows luxuriantly in ordinary nutrient gelatine at 15° C. (59° F.), not fluidifying. On potato it grows vigorously, resembling the pneumococcus of Friedländer. He claims as the result of inoculations of pigeons, rabbits, and white mice, that he produces a disease which is usually fatal; that a pseudo-membrane forms in the trachea; and that the bacteria are found in the blood and in the internal organs, especially in the kidneys. Important as these results of Emmerich may seem to be, they have not been confirmed by others, and in several particulars there appear to be such possibilities of serious error in his technical procedures that it would seem unwise to attach a definite value to them until fuller detail or control research by others has given us more positive data. At a later period Emmerich (5), while still insisting upon the importance of his alleged diphtheria bacillus, calls attention to the frequent occurrence of streptococci in the diphtheritic membranes.

In 1886, Babes (6) reports the cultivation from six cases of pharyngeal diphtheria, from eight cases of croupous laryngitis, from three cases of diphtheria after measles, from one case of diphtheria after scarlatina, and from one case of diphtheria of the conjunctiva, of the bacillus of Loeffler among others. In these cases he found streptococci associated with Loeffler's bacillus. The details of his work are not given in this report nor is there any intimation that he tested the pathogenic properties of the isolated bacilli. In view of the incomplete character of this work and of the later discovery (see below) by Loeffler and v. Hoffmann, of a somewhat similar non-virulent bacillus, these results of Babes cannot be given much weight as evidence of the frequency of the occurrence or importance of Loeffler's bacillus in diphtheria.

In April, 1887, Loeffler (7) again made a communication upon the subject, in which he states that in ten cases in which fresh diphtheritic

membranes were examined he found the bacilli in all. He also describes the occurrence in one of his cases of a bacillus very similar in its morphological and biological characters to those found in his earlier studies, but which was without effect upon animals. This he calls the "pseudo-diphtheritic bacillus."

Penzoldt (8), in 1887, as the result of a long series of somewhat rambling experiments found that he could not succeed in inducing typical diphtheria in rabbits, chickens, or pigeons by inoculation either with fresh diphtheritic membranes or with cultures from them. He sometimes obtained thick or thin, for the most part loose, pellicles over the inoculated surfaces, but these could be induced by a variety of bacteria which obviously have nothing to do with diphtheria. He gives but little detail as to the species which he isolated by culture. He emerges from his researches with a disposition to believe that diphtheria, in a clinical sense, may be due to a variety of causes.

Fränkel, in the same year, described a case, in an adult, of diphtheria of the fauces and larynx with secondary phlegmonous inflammation and pleurisy and pericarditis, from which he cultivated the streptococcus pyogenes (9). He describes another case, also an adult, of diphtheria of the pharynx and larynx with secondary ulcerative endocarditis From the pseudo-membranes as well as from the heart lesion he cultivated the streptococcus pyogenes.

D'Espine (10) has reported the isolation of a bacillus similar to that described by Loeffler, but the meagreness of his published data prevents a definite judgment as to the importance of his observations.

Early in 1888 v. Hoffmann (11) described the very frequent occurrence —in twenty-six out of forty-five cases—on the mucous membrane of the larynx, as well in diphtheria, scarlatina, measles, and simple catarrh as in quite normal conditions, of a bacillus apparently identical with the pseudo-diphtheritic bacillus of Loeffler. That is, it was very similar in morphology and growth to the diphtheria bacillus of Loeffler, but not virulent. A careful comparison of the genuine with the pseudo-diphtheritic bacillus led v. Hoffmann to the recognition of biological and morphological distinctions by which the two forms could be recognized. With this knowledge he again set to work, and from eight new cases of diphtheria he isolated the genuine Loeffler bacilli. But these, in one case, were not at all virulent for guinea-pigs, and in the others in a much less degree than those originally isolated by Loeffler. V. Hoffmann further isolated from three cases of measles the bacillus of Loeffler, the cultures in one case being virulent to guinea-pigs, in two not. Six times in nineteen cases of scarlatina he found Loeffler's bacilli, and these in no case were virulent. From the pharynx or larynx in eleven healthy cases he isolated Loeffler's bacillus four times; one of the cultures was virulent, the other not. V. Hoffmann seems to have made no animal

experiments with the view of proving the etiological relation to diphtheria of these, Loeffler's bacilli, which he isolated. He simply tested their capacity of killing guinea-pigs by subcutaneous inoculations.

Recently, Roux and Yersin (17) have examined, partly by culture methods and partly morphologically, fifteen (?) cases of diphtheria, and state that they found the Klebs-Loeffler bacilli in all. The record of their observations is so incomplete, however, that it does not appear whether or not they made cultures from all the cases. They apparently did not make use of the plate-culture method, but sowed the material from the pseudo-membranes directly upon tubes. Animal experiments were made, but were, so far as appears, comparatively few in number and apparently with cultures from but a few of the cases. These, in general, are said to be confirmatory of Loeffler's results. But as the local results of inoculations on injured mucous membranes are said by the authors, without detailed description, simply to be "false membranes," it is difficult to judge of their nature. The most suggestive part of their observations is the induction of symptoms of paralysis in animals by the injection of the fluids in which the bacilli had grown, and which had been freed from the germs by filtration. What bacteria, if any, other than those which they fixed upon as similar to the species isolated by Loeffler, developed in their cultures, does not appear, nor are such details of technique given as would inspire the reader with confidence that the cultures with which the animals were inoculated were actually pure cultures.

On the whole, the observations and experiments of these writers appear to be confirmatory of the work of Loeffler. But the records are so incomplete, and their technical procedures are of such a questionable nature and are so meagrely described, that it is very difficult to conclude whether the work is really of importance or not.

Since the perfection of the methods of staining bacteria in the tissues, a number of more or less careful simple morphological studies of diphtheritic lesions have been made, with the general result of showing what indeed had been demonstrated, even with the crude facilities of the older technique—that the diphtheritic membrane contains, as a rule, large numbers of bacteria of various shapes—the micrococcus form generally preponderating. This method also shows that the bacteria are sometimes present in the living tissue immediately about the seat of the false membrane, when this is formed, and that they sometimes are and sometimes are not to be found in the blood and internal organs.

If now we make a close summary of the actual results of the recent experimental work on diphtheria along the new lines of research, rejecting all simple morphological studies as incomplete and inconclusive, and confining ourselves to those cases only in which the culture methods have definitely revealed the identity of the bacteria, we find that from 16 cases of diphtheria out of 20 Loeffler isolated a bacillus which was capable of

killing certain of the lower animals when introduced subcutaneously, and which, as tested from 6 cases when inoculated into a wounded mucous membrane, might induce an inflammation with a fibrinous pellicle. V. Hoffmann isolated the bacilli of Loeffler from 8 cases of diphtheria, but he cultivated them from 13 out of 33 cases of healthy and diseased air-passages in which there was no diphtheria present. Thus, while the Klebs-Loeffler bacilli have been cultivated 24 times from 28 cases thus examined, they have been found in 14 out of 34 cases which had nothing to do with diphtheria.

The results of animal inoculations with the pure cultures of Loeffler's bacillus are, on the whole, rather less convincing than the simple morphological and biological results. Certain animals are apt to die as the result of the inoculation of the Klebs-Loeffler bacillus, and inoculation on an injured mucous membrane may be followed by the formation of a fibrinous pellicle which somewhat, but not very closely, resembles the pseudo-membrane common in diphtheria. But if we recall how many agencies there are capable of producing a pseudo-membrane on the mucous surfaces, and how many forms of bacteria kill animals on subcutaneous injection with symptoms and lesions very similar indeed to those induced by Loeffler's bacillus, it seems to the writer that the reasons for identifying the animal with the human disease are neither definite nor convincing.

At present, then, it would seem that actual existing knowledge justifies us only in the conjecture that diphtheria is caused by some form or forms of bacteria. What the species is, or whether there are more than one, we do not know. We are confronted to-day with the same series of questions about which discussion has centred for many years. Are the diseases called pseudo-membranous pharyngitis and pseudo-membranous laryngitis or simple croup really, after all, identical with genuine diphtheria, differing only in that in one case there is a simple local lesion, while in the other there is a general systemic infection? It does not help us much in studying the etiology to assume that the systemic infection which stamps diphtheria as an acute infectious disease, and gives it clinically a more or less definitely isolated position, is the crucial test, because it is not only quite possible, but highly probable, that here, as for example in a poisoned wound of the finger, the process may in one set of cases be and remain local, while in another it may be local for a time and then become general, or be general almost from the outset.

That there is a form of croupous inflammation of the pharynx, larynx, and other air-passages, not due to active chemical irritants, and not occurring, as we say, "spontaneously," which is not obviously infectious and not apparently communicable from one individual to another, would seem to be well established by clinical observations. On

the other hand, there is every reason to believe that many cases presenting the clinical features of simple croupous inflammation may be really diphtheria—as judged from the standpoint of their etiology—but in which, from some ineffective condition of the germs, from the smallness of the numbers inoculated, or from the resisting capacity of the individual, the action of the poison is limited to the seat of inoculation. We are constrained still to query whether the diphtheritic inflammation of the pharynx and other air-passages which so frequently accompanies scarlet fever and measles belongs to a different group from that which is not thus associated.

Character and Scope of Observations, Technique, etc.

The writer does not set before himself, in this paper, so ambitious a purpose as the complete solution of this problem of the etiology of diphtheria, which is one of the most complex and difficult, as it is one of the most important, questions in experimental pathology. But he wishes simply to record a series of observations made, in the light of modern science and with improved technical facilities, on a number of cases of diphtheria occurring in and near New York. When from one and another observer a sufficient number of such sets of cases shall have been studied and recorded, it is probable that then, and not until then, shall we be able to state definitely what the disease really is, the etiology of which we are seeking to discover. The problem is, at best, one of extraordinary difficulty, because we are liable to have on the exposed parts most often the seat of the local lesion of diphtheria the greatest variety of living bacteria, from the air, from water, and from food. These must all be carefully studied one by one, in each particular case, with all the precautions against error which experience and patience can furnish to the investigator.

The cases thus fully studied were those in which the clinical history left no room for doubt as to the diagnosis; all the fatal cases of extensive pseudo-membranous laryngitis, as well as pharyngitis, being regarded as diphtheria. They were most of them in children, were fatal, and early autopsies were made in nearly every case. The autopsies in most of the cases were made by a most competent and experienced observer—Dr. William P. Northrup—to whose zeal and courtesy I am indebted for much of the material. The parts to be subjected to examination in most of the cases were placed in carefully cleansed jars immediately on removal from the body, and brought or sent at once to the laboratory, where the detailed biological examination was begun without delay and carried on with the use of all the precautions against accidental contamination which the technical procedures formulated by Koch so admirably cover.

It has seemed to the writer wise, in undertaking these studies, not to

seek to gather material for observation from a great variety of sources and from persons whose illnesses had run their course under the greatest variety of conditions, but to limit it largely to children, and especially to children which are placed under the general sanitary conditions prevalent in children's hospitals and asylums, where the cases may be closely observed from the outset and the surrounding conditions studied and more or less controlled.

Thus, while most of the cases came from one large foundling asylum,[1] in which the sanitary conditions are rather above the average in such institutions, in order to have some measure of comparison other cases were taken from other similar public institutions in other parts of New York City, and two cases occurred in private practice in one family in an adjacent town.

It will be readily appreciated, by those familiar with the details of such researches as these, that such a limitation of the problem is rendered imperative by the large expenditure of time which is required for the working out in full of any single case. Thus it was that a considerable amount of material from a number of cases of diphtheria (membranes, etc.), which was kindly placed by his colleague at the writer's disposal, was not made use of at all, partly because of the lack of time and partly because it was believed that a small group of cases fully worked out might lead to more reliable and tangible results than many only partially studied. In order, however, to avoid a misunderstanding of this selection of cases, it should be stated that material from every case of diphtheria which belonged in the group above indicated, and which came in proper condition for study into the writer's hands, was worked carefully through, and the results of all are recorded below.[2]

The primary task which the writer set before himself in the examination of these cases of diphtheria was, first, to ascertain, by the modern culture methods, what living microörganisms, if any, were present at and about the seat of the lesion or in the internal organs with sufficient frequency to justify the assumption that they might stand in an etiological relationship to the disease; to separate these in pure cultures, and study their life-history. Second, to learn by morphological examination of sections of the hardened and stained tissues what were the situation, distribution, and, so far as might be estimated, numbers of the microorganisms which might be present. Third, to ascertain the effects upon animals of the inoculation of the isolated species.

It seems hardly necessary to say that in all cases plate cultures were

[1] These studies have extended over a period of two years, so that the cases from the large foundling asylum represent two distinct epidemics in successive years.

[2] I wish especially to acknowledge my indebtedness for material to Drs. J. Lewis Smith, R. Van Santvoord, L. Putzel, C. W. Oakes, and Mr. Charles Laylbach.

first made, and that these were manipulated in the usual way for the separation of individual species. Gelatine, agar, agar-glycerine, and blood-serum plates were used—the agar and agar-glycerine being the most generally efficient. Save for the gelatine cultures, the temperature of 37° C. (98.6° F.) was uniformly employed for the growth. The plate-cultures were mostly made either in shallow, covered dishes or in Esmarch tubes—generally in the former. Duplicate plates were in all cases made for the purpose of control, not only in the different cases, but for each part or organ subjected to examination. With this large number of plates, it was possible to make the sowings so dilute that the very large number of bacteria usually present in material from the air-passages could be readily obtained in isolated colonies.

It seemed unnecessary for the purpose in view, except in the first cases, to do more than isolate the similar species which developed in the different cases. Thus, a large number of forms which were familiar to the writer as occurring in the air, or water, or food, or those which control-cultures from the mouths and throats of healthy children, or those not suffering from diphtheria, showed to be common and unimportant, could be ignored after such observations on their characters as sufficed for their identification. Single colonies of different species developing on the plates were also for the most part ignored in the examination, since the probabilities were greatly against such isolated forms being of any significance. The task was thus narrowed down to the investigation of those forms of bacteria which developed in considerable numbers on the plates, and especially those similar forms which were found to occur on the plates made from different cases.

The colonies of these forms which were to be studied in detail were transferred first to a second set of plates and these, if evidently pure, were inoculated into tubes. In some cases, where large numbers of forms were present, several successive transfers from plate to plate were necessary to insure purity. The examination of the plates was made in the culture-room of the laboratory, which is kept scrupulously clean by the frequent washing of its shelves and floor with disinfecting solutions, and the air-supply to which, driven in by the ventilating fans, is filtered through frequently changed layers of dry cotton. In this way the air of the culture-room is so thoroughly free from germs that aërial contamination of the exposed plates during their examination was very infrequent. Thus it was possible to follow the development of colonies on the plates, even though they might be slowly growing forms, for long periods without danger of overgrowth by adventitious contaminations.

The writer had, fortunately, been able to acquire practical familiarity with the morphological and biological characters of Loeffler's bacillus from cultures which were the direct descendants of those isolated by Loeffler himself, and brought directly from Berlin.

Large numbers of duplicate sections from the hardened tissues of all the cases were prepared, and studied with a Zeiss $\frac{1}{12}$ homogeneous immersion lens and the Abbé condenser. Some of these sections were stained with Loeffler's alkaline methylene-blue solution, some by Gram's method, and some with the simple aqueous fuchsin solution. By the use of these three methods of staining in each case, the possibility of overlooking any form of bacteria in the tissues examined was largely reduced.

Having thus indicated the scope of the work and the precautions against error, it does not seem necessary to give an account of each case in detail, but to present the results in the form of a *résumé*.

The cases of diphtheria studied were twenty-four in number.[1]

Cases of Diphtheria Examined.

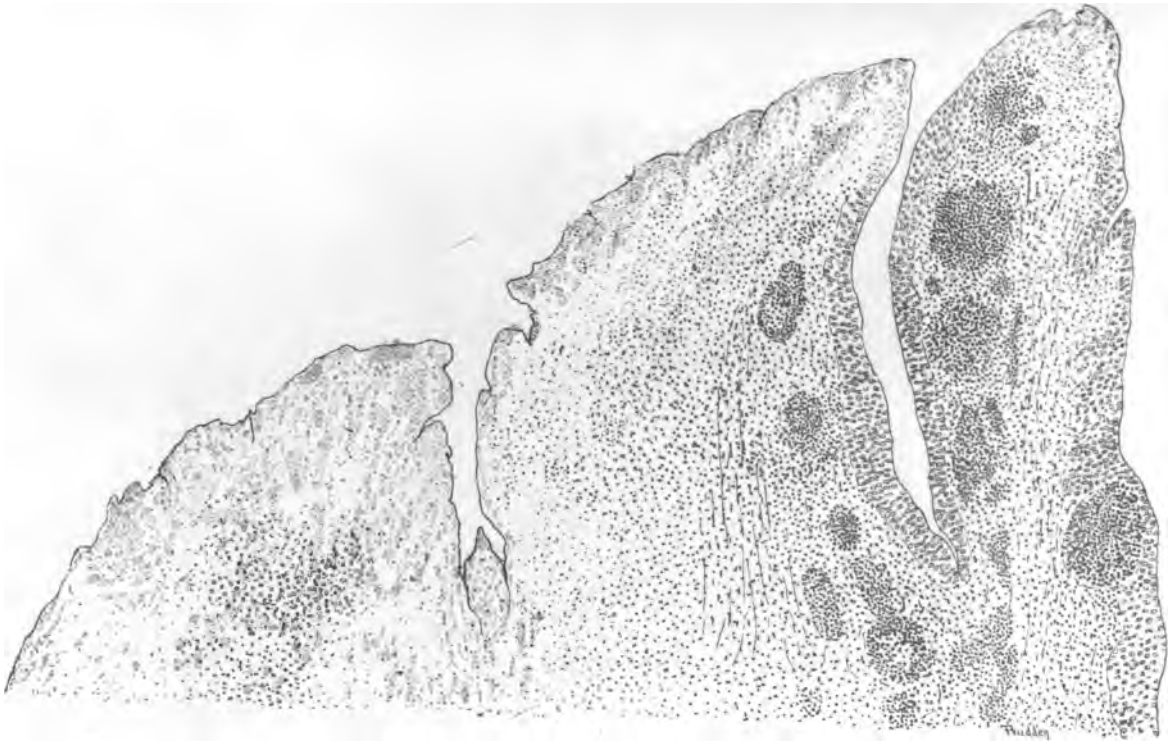
Case I.—*Clinical history:* Male, seven months. Feb. 8th, pertussis; 10th, diphtheria; 12th, intubation; 14th, death. *Autopsy*, twelve hours: Moderately thick and softening membrane in the trachea extending into the larger bronchi; tracheal wall at the lower end of tube eroded; broncho-pneumonia. *Bacterial examination—Morphological:* Enormous numbers of cocci, single, clustered, and in longer and shorter chains in all parts of the false membrane, particularly in its deeper layers; a few scattering, large, plump bacilli in the membrane. *Cultures: Streptococci*[2] in enormous numbers. Staphylococcus pyogenes aureus. A few scattering forms, the most abundant of which was a large bacillus resembling bacillus subtilis. In the false membrane in the trachea, the streptococcus exceeded the staphylococcus in numbers in about the proportion of 1300 to one, while in the bronchial exudate the proportion was about ten to one.

Case II.—*Clinical history:* Male, two years and seven months. Jan. 28th, pertussis; 29th, acute tonsillitis, temperature 105°; 30th, diphtheria, albuminuria. Feb. 2d, broncho-pneumonia; 15th, death. *Autopsy*, three hours: Dense membrane in the lower part of trachea and in the larger and smaller bronchi; broncho-pneumonia in both lower lobes; parenchymatous nephritis. *Bacterial examination—Morphological:* In the bronchial exudate, especially in its deeper layers, were large numbers of cocci scattered in clumps and in longer and shorter chains. Similar cocci were found between the necrotic epithelial cells of the mucous membrane. A few chains of cocci were present in the lymph spaces of the mucosa and submucosa. A few stout bacilli were scattered through the false membrane. *Cultures: Streptococci* in large numbers. Staphylococcus pyogenes aureus. Aside from a few scattering colonies of variously shaped bacteria there were considerable numbers of a common white fluidifying yeast.

[1] The writer has made a large number of simple morphological examinations of sections of the false membranes and underlying parts from other cases of diphtheria than those now to be recorded; and, while it may be said of these that the cocci are, in almost every case, the prevailing form of bacteria, yet, as the morphological examination does not suffice to identify the species, the observations serve as contributing evidence of such moderate value, in the present state of our knowledge, that it does not seem worth while to do more than merely mention them here.

[2] For the sake of convenience, the morphological and biological character of this streptococcus will be given in detail below.

PLATE I.



Portion of tonsil in diphtheria (Case VI.) showing infiltration of the tissues with streptococci, with necrotic tissue in the vicinity of the bacteria. A small portion of the invaded tissue, more highly magnified, is shown in Plate II., Fig. 3.

CASE III.—*Clinical history:* Umbilical phlegmon developed seven days after birth. At the same time pseudo-membrane was noticed on the pharynx, and there was considerable purulent conjunctivitis. With an increase in the amount of pseudo-membrane, and more intense phlegmonous inflammation about the umbilicus, the child died fourteen days after birth.[1] *Autopsy:* Dense firm pseudo-membrane over both tonsils and epiglottis; voluminous softening membrane in trachea and larger bronchi; broncho-pneumonia in both lower lobes with pulmonary congestion; parenchymatous degeneration of kidney; extensive umbilical phlegmon with suppuration. *Bacterial examination—Morphological:* Throughout the entire extent of the false membrane great numbers of cocci scattered and in chains. These were present as well in the deeper as in the superficial portions of the membrane. A small number were found scattered in the lymph spaces of the submucosa. A few scattering large bacilli were found in the membrane. *Cultures: Streptococci* in enormous numbers. Staphylococci pyogenes aureus and albus. A considerable number of large, long and plump, and short plump, round-ended bacilli. Cultures from the phlegmon showed both the streptococci and staphylococci.

CASE IV.—*Clinical history:* Female, three years and eight months. April 28th, varicella; 29th, croupy; 30th, dyspnœa and cyanosis, temperature 104.8°. May 2d, broncho-pneumonia, temperature 105.5°; 4th, death. *Autopsy,* twelve hours: Abundant pseudo-membrane in pharynx, larynx, trachea, and larger bronchi, firm and closely adherent above, and softening in the trachea; broncho-pneumonia in both lower lobes; parenchymatous degeneration in kidneys. *Bacterial examination—Morphological:* Moderate numbers of cocci scattered and in longer and shorter chains in the false membrane. *Cultures: Streptococci* in enormous numbers and in nearly pure culture. A few staphylococci pyogenes aureus and a few other scattering forms on some of the plates.

CASE V.—*Clinical history:* Female, two years and eight months. May 5th, had been two weeks convalescent from scarlet fever when a pseudo-membrane developed on the soft palate. Temperature 101°; albumin 80 per cent., granular casts. Two days later died, apparently from heart failure. *Autopsy,* six hours: Very slightly developed membrane, best formed in the trachea; moderate pulmonary congestion. *Bacterial examination—Morphological:* In the tracheal exudate a small number of cocci and a few stumpy bacilli. *Cultures: Streptococcus* colonies developed in small numbers on the plates, with a few colonies of staphylococcus pyogenes aureus and a few scattering forms.

CASE VI.—*Clinical history:* Female, three years. May 10th, pharynx red; 11th, scarlatina; 15th, pseudo-membrane in pharynx; 17th, diarrhœa; 20th, croupy cough, temperature 103°; 23d, weaker, dyspnœa; 25th, intubation, death. *Autopsy,* three hours: Firm pseudo-membrane over both ulcerated tonsils. Pseudo-membrane in upper portions of trachea. Commencing broncho-pneumonia. Kidneys congested; parenchymatous degeneration. *Bacterial examination—Morphological:* The pseudo-membrane over the surface of the tonsils and the necrotic tissue on the tonsils near the ulcers and in the false membrane were crammed with cocci, single, massed, and in long and short chains. (See Plate I.

[1] This case was, in part, reported by Dr. J. Lewis Smith to the New York Pathological Society, June 13, 1888.

and Plate II., Fig. 3.) The lymph nodules or follicles of the tonsils contained numerous single and chain cocci scattered between the cells. No bacteria were found by morphological examination in the kidney. *Cultures: Streptococci* were present in enormous numbers in the cultures from the pseudo-membrane of the tonsils and the necrotic tissue beneath it. They were also very abundant in the membrane from the upper portion of the trachea, less abundant in the softening portions below. Streptococci in pure culture and in considerable numbers from the kidney. Staphylococcus pyogenes aureus, most abundant in pharyngeal membrane.

CASE VII.—*Clinical history:* Male, two and a half years. May 24th, vomiting and croupy cough; 25th, pseudo-membrane in pharynx; 26th, temperature 102½°, croupy cough increasing, dyspnœa, death. *Autopsy*, thirty hours: Dense voluminous pseudo-membrane over both slightly roughened and ulcerated tonsils, over the entire larynx, and extending down to the larger bronchi; membrane softened below; commencing broncho-pneumonia. *Bacterial examination—Morphological:* In the pseudo-membrane over the tonsils, in the crypts of the tonsils, and in the necrotic tissue at the edges of the tonsillar ulcers were large numbers of cocci scattered and in short chains. A few stout bacilli in the superficial portions of the membrane. *Cultures: Streptococcus* colonies in enormous numbers in cultures from all parts of the membrane. Staphylococci pyogenes aureus and considerable number of scattering forms. Kidney cultures showed considerable number of streptococcus colonies, and nothing else.

CASE VIII.—*Clinical history:* Female, one year. Three days before death follicular stomatitis; during next night restlessness; then collapse and death. *Autopsy*, two and a half hours: Tonsils ulcerated and covered with firm pseudo-membrane; dense, firmly adherent membrane on the posterior pharynx, upper part of trachea, and œsophagus; enterocolitis; parenchymatous degeneration of kidney; commencing broncho-pneumonia. *Bacterial examination—Morphological:* Everywhere in the membrane and in the necrotic tissue near the ulcers of the tonsils were great numbers of cocci, scattered, clustered, and in large and short chains. These streptococci were less abundant in the tracheal membrane than above. Few scattering forms of bacilli. *Cultures: Streptococcus* colonies developed in vast numbers from the pseudo-membrane. Kidney cultures were negative, while from the spleen pure cultures of streptococci grew. Staphylococci pyogenes albus in small numbers grew from the membranes, while there were large numbers of the common "chain bacillus" of the mouth and a few scattering forms.

CASE IX.—*Clinical history:* Female, two years. May 27th, pneumonia, left side; temperature 106°. Seemed to recover. June 4th, pseudo-membrane on left tonsil. 5th, croupy cough, dyspnœa; intubation; broncho-pneumonia developed. 6th, death. *Autopsy*, twenty-one hours: Softening membrane over both tonsils; broncho-pneumonia, with pleurisy in left lower lobe. *Bacterial examination—Morphological:* In the membrane on tonsil considerable numbers of scattering cocci and streptococci; nothing else. *Cultures: Streptococcus* colonies grew in considerable numbers from the membrane; also large numbers of staphylococci pyogenes aureus, considerable numbers of fluorescent bacilli (doubtless from water) and of the "chain bacillus."

CASE X.—*Clinical history:* Male, three years. May 7th, scarlatina;

PLATE II.

FIG. 1.—Streptococcus diphtheriæ; beef-tea culture:—showing the tendency of the bacteria to group themselves in pairs as well as in chains.

FIG. 2.—Edge of streptococcus colony growing on the surface of an agar-plate:—showing the loops and chains which grow out from the central denser portion of the colony.

FIG. 3.—A small portion, more highly magnified, of the tissue of the tonsil shown in Plate I., invaded by the streptococcus.

11th, pseudo-membrane on tonsils; 14th, otitis; 16th, swelling under left jaw. June 1st, abscess opened behind tonsil; 7th, death. *Autopsy*, four hours: Pseudo-membrane dense and firmly adherent at the base of tongue and over both tonsils, which were ulcerated. The membrane extended over the epiglottis, completely investing the larynx to below the vocal cords, where it became softened and shreddy. Chronic and commencing fresh broncho-pneumonia. Bronchial lymph glands greatly swollen and red. Kidneys, parenchymatous degeneration. *Bacterial examination—Morphological:* Everywhere in the pseudo-membrane and in the necrotic mucosa beneath were vast numbers of cocci, singly, clustered, and in long and short chains. A few isolated clusters of moderately stout, long bacilli were found near the surface of the pseudo-membrane. The lymph sinuses of the bronchial glands were distended, and contained an abundant network of fibrin. No bacteria were found in the spleen, kidney, liver, or bronchial lymph glands. *Cultures: Streptococci* in great numbers developed from various parts of the pseudo-membrane. The spleen cultures were negative, but a few colonies of streptococci, and nothing else, grew from the kidney and liver. Staphylococci pyogenes aureus grew in considerable numbers from the membrane, and a few from the liver. Some scattering forms of colonies developed from the membrane, the most abundant of which were the "chain bacillus" and a fluorescent water bacillus.

Case XI.—*Clinical history:* Female, three years and two months. Feb. 1st, rectal abscess; 5th, croupy cough; 6th, intubation; 7th, death. *Autopsy*, sixteen hours: Pseudo-membrane in larynx, trachea, and larger bronchi. *Bacterial examination—Morphological:* Moderate numbers of cocci, mostly in pairs, in the pseudo-membrane. *Cultures: Streptococcus* colonies grew from the membrane in nearly pure culture. Staphylococci pyogenes aureus developed in small numbers, and a few scattering forms.

Case XII.—*Clinical history:* Female, two and a half years. Feb. 9th, croupy cough, pseudo-membrane; 11th, intubation, broncho-pneumonia; 14th, temperature 102½°; 16th, death. *Autopsy*, two hours: Pseudo-membrane in pharynx, larynx, and trachea; ulcer in trachea at end of tube; broncho-pneumonia in both lower lobes. *Bacterial examination—Morphological:* Large numbers of cocci, many in chains, in the pseudo-membrane; few scattered bacilli. *Cultures: Streptococcus* colonies in considerable numbers grew from the membrane. Also staphylococcus pyogenes aureus and a few scattering colonies of stout, large bacilli.

Case XIII.—*Clinical history:* Female, two years and two months. Brought to the hospital with vulvitis, which developed into erysipelas about vulva. Eight days later, false membrane appeared on the tongue; then croup followed, with death in five days. *Autopsy*, two hours: Vulva denuded of epithelium, dusky red. Dense, firm, closely adherent membrane on velum and uvula; tonsils ulcerated and covered with false membrane, which extended down over the posterior surface of the epiglottis and onward to just below the vocal cords; no broncho-pneumonia. *Bacterial examination—Morphological:* Large numbers of cocci, single, in pairs and chains, in the deeper layers of pseudo-membrane, and extending deeply into the underlying tissue, accompanied here, when in large numbers, by necrosis. Other scattering forms were abundant. Scattered clusters of slender bacilli were found in the pseudo-

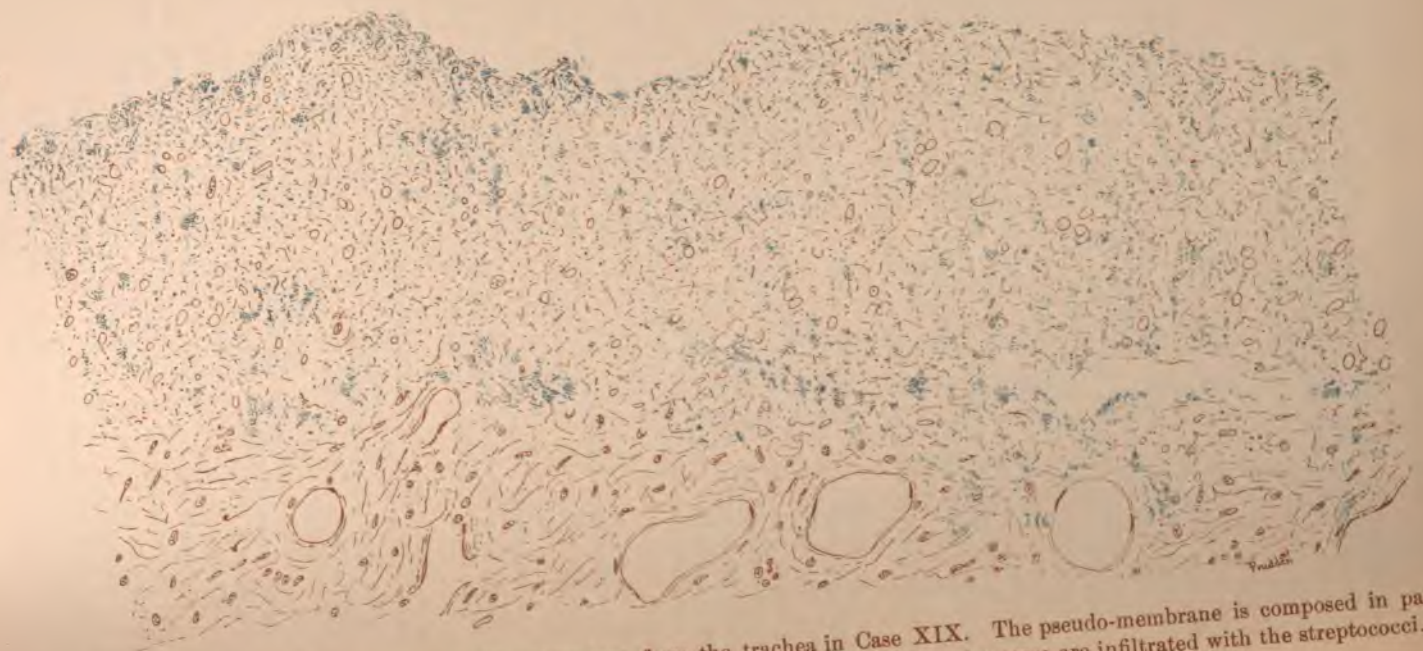
membrane near the vocal cords, especially in the most superficial portions. *Cultures:* In the pseudo-membrane from the tonsils, pharynx, and larynx, enormous numbers of *streptococci* in nearly pure cultures; a few staphylococci pyogenes aureus and a few scattering forms, mostly large bacilli.

Case XIV.—*Clinical history:* Female, two months. Returned to hospital from out-nursing with bad cough, and after being for eight days in room with considerable number of children, sick and well, developed diphtheria, rapidly followed by broncho-pneumonia, with death in two days. *Autopsy*, eleven hours: Pseudo-membrane over both tonsils; on posterior pharynx and in larynx and trachea down to bifurcation membrane softening. *Bacterial examination—Morphological:* The pseudo-membrane had in many places considerably involved the superficial portions of the mucosa. Moderate numbers of cocci and streptococci in the pseudo-membrane, together with a considerable number of stout, long bacilli in the more superficial portions. *Cultures:* False membrane on tonsils, pharynx, and larynx all alike showed large numbers of *streptococci*. There were also many staphylococci pyogenes aureus and a considerable number of colonies of coarse, stumpy bacilli.

Case XV.—*Clinical history:* Male, three years. Returned to hospital Dec. 3d. On Dec. 13th developed diphtheria; 20th, intubation; 23d, broncho-pneumonia, cyanosis, death. *Autopsy*, six hours: Tonsils swollen, not ulcerated. Small patch of pseudo-membrane on pharynx, and extending from the tip of the epiglottis to the medium bronchi, firm and closely adherent above. Congestion and commencing broncho-pneumonia in both posterior lower lobes. Cortex of kidney thick and opaque, spleen and liver appear normal. *Bacterial examination—Morphological:* Surfaces of tonsils and crypts intact, with small masses of diplococci and streptococci here and there in the crypts. In the pseudo-membrane enormous numbers of cocci in pairs and chains, especially in the deeper layers and in the necrotic tissue of the submucosa beneath. Other scattering forms were present, but none abundant. No bacteria found in the viscera. *Cultures:* Large numbers of *streptococcus* colonies developed from scrapings of tonsil crypts, from various parts of the pseudo-membrane, and from the lungs. Cultures of liver, kidney, and spleen were negative.

Case XVI.—*Clinical history:* Male, three and a half years. Six months in hospital. Dec. 20th, croupy cough, relieved by emetic. 21st, croup symptoms continued; no pseudo-membrane seen on pharynx. 22d, died. *Autopsy*, four hours: Tonsils swollen with large pit-like crypts. Dense, firm pseudo-membrane extends from tip of epiglottis down to bifurcation of trachea; firmly attached as far down as vocal cords, below this loose. Bronchitis, but no broncho-pneumonia. Bronchial and tracheal lymph glands swollen and red. Kidneys, parenchymatous degeneration. Ulcerative gastritis and ileo-colitis. *Bacterial examination—Morphological:* In the pseudo-membrane very few scattered bacteria were found. There were a few cocci, mostly on the surface, and a few short, stout bacilli. There was no necrosis of the tonsils, and but few bacteria in the crypts. No bacteria of any form were found in either bronchial or lymph glands, or in the kidney, liver, or spleen. *Cultures:* In the pseudo-membrane and tonsil crypts were found a considerable number of short, stout, round-end bacilli and sev-

PLATE III.



Section of diphtheritic pseudo-membrane with submucosa, from the trachea in Case XIX. The pseudo-membrane is composed in part of inflammatory exudate, in part of necrotic mucous membrane, and both this and the submucosa are infiltrated with the streptococci.

eral scattering forms. No streptococcus colonies developed. Cultures from the lymph glands and viscera were sterile.

CASE XVII.—*Clinical history* is wanting, save that the child, with several others in the same hospital, was suffering from measles when the diphtheria developed. No pseudo-membrane was seen in the pharynx, and the child died soon after the diphtheritic symptoms manifested themselves. *Autopsy:* Tonsils much swollen, with somewhat ragged surfaces. Voluminous, firm pseudo-membrane from the tip of epiglottis down to medium bronchi, loosened and softening below. Commencing broncho-pneumonia. Cortex of kidney slightly thickened, liver and spleen appeared normal. *Bacterial examination—Morphological:* Large numbers of cocci, streptococci, and diplococci in tonsil crypts and over the eroded surfaces. Similar forms in moderate numbers in the deeper layers of the pseudo-membrane. Aside from these, only scattering forms. No bacteria found in kidney, liver, or spleen. *Cultures:* Large numbers of *streptococci* from all parts of the pseudo-membrane and from the tonsil crypts, also some scattering forms. A few streptococcus colonies developed from the kidney and spleen; liver cultures were negative.

CASE XVIII.—*Clinical history:* Child, one year. Had been in hospital for six months with bronchitis. Measles developed on Dec. 24th, and ran a course of ordinary severity; croupy symptoms began early in the disease, and on Jan. 8th diphtheritic symptoms were pronounced, though there was no pseudo-membrane on the pharynx. The child died on Jan. 9th. *Autopsy:* No pseudo-membrane on pharynx, but on the larynx below the epiglottis were numerous, small, irregular, firm, closely adherent patches of pseudo-membrane; the tracheal and bronchial glands were red and swollen. Viscera not examined. *Bacterial examination—Morphological:* Large numbers of cocci, single, paired, and in chains, were irregularly grouped, especially in the deeper layers of the pseudo-membrane and sparsely infiltrated the mucosa beneath. No bacteria were found in the tracheal and bronchial glands. *Cultures:* Small numbers of various scattering forms, among which a few staphylococci pyogenes aureus were present. The *streptococcus* was present in large numbers.

CASE XIX.—*Clinical history:* Child, one year and eight months. Four months in hospital with diarrhœa. The child was attacked with measles of a severe form, with high temperature, vomiting, and cough. Croupy symptoms appeared on the third day, and increased steadily till the sixth, when tracheotomy was performed. Slight improvement followed, but the child died thirty-six hours after the operation. *Autopsy:* Small particles of pseudo-membrane on tonsils. Dense voluminous membrane in larynx and trachea. Cheesy bronchial glands, with localized tubercular inflammation of the lungs, and fresh broncho-pneumonia. *Bacterial examination—Morphological:* Pseudo-membrane closely adherent in places with much involvement of the epithelium, and superficial necrosis of the submucosa. Enormous numbers of cocci, single, in pairs and chains, in the deeper layers of the pseudo-membrane, in the necrotic mucosa, and infiltrating in smaller numbers the lymph spaces of the submucosa (see Plate III.). No bacteria found in the lymph glands. *Cultures:* From the pseudo-membrane of the larynx and trachea were cultivated large numbers of *streptococci*. Besides these a few colonies of staphylococci pyogenes aureus and albus, and a few scattering forms.

CASE XX.—*Clinical history:* Child, two years. Twenty-three days in

hospital with diarrhœa. Measles developed, running a severe course with high temperature. A croupy cough developed early with dyspnœa and symptoms of pneumonia. On the eighth day intubation was practised without benefit, and the child died in a few hours. *Autopsy:* Tonsils were swollen; the mouth of the crypts dilated. No false membrane was found, but both laryngeal and pharyngeal mucous membranes were swollen and intensely red. The bronchial glands were red and swollen. The lungs were congested. *Bacterial examination—Morphological:* The epithelium covering the surfaces of the tonsils and lining the crypts was for the most part intact, but here and there in the depth of the crypts small patches of necrotic epithelium were found, which were infiltrated with streptococci. A few scattering cocci and streptococci were found on the surface of the intact epithelium in the crypts. In the ventricles of the larynx and about the rima glottidis were considerable numbers of cocci and streptococci, with a few stout bacilli. The epithelium in the ventricles of the larynx was in places partially detached and necrotic, and in these regions numerous streptococci were found beneath the epithelium, and in the superficial layers of the submucosa. In this case, then, in which there was no pseudo-membrane, in two places, at least, there appeared to be a commencing invasion of the tissues with streptococci. No bacteria were found in the lymph glands. The air-spaces of the lungs contained enormous numbers of streptococci and cocci. *Cultures:* Scrapings from the tonsil crypts and from the region of the vocal cords showed considerable numbers of *streptococci*, together with scattering forms.

Case XXI.—*Clinical history:* Child, one year and six months. In hospital six months. Measles running a severe course, with temperature 104°. Dyspnœa was developed early, and the child died twenty-four hours after the commencement of the disease. *Autopsy:* Tonsils were swollen and ragged. In the pharynx, at the base of the epiglottis, on either side, was a small, irregular patch of pseudo-membrane, dense and closely adherent. Small white-surfaced ulcers were found just below the rima glottidis. No pseudo-membrane visible elsewhere, but the larynx was of a dusky red color. *Bacterial examination—Morphological:* The bloodvessels of the mucous membrane were widely dilated. In the small patches of pseudo-membrane in the pharynx, as well as at the seat of the ulcer, below the rima glottidis, there were large collections of cocci, mostly in short chains, which penetrated the submucosa, and on either side extended beneath the intact epithelium. In the vicinity of these regions, but especially in the pharynx, there were considerable collections of cocci and streptococci lying closely packed against the surface, or infiltrating the superficial epithelial layers. In the regions where these bacteria were numerous the epithelium was either necrotic or was much thickened and peeling off in large masses. The tonsils were intact, but in the mouths of the crypts, as well as entangled in the mucus of the larynx, were a few cocci, single or in short chains, and a few scattering forms of other bacteria. In this case we seem to have the commencement of the formation of the pseudo-membrane, and the invasion of the adjacent tissues with streptococci. *Cultures:* A moderate number of *streptococci* grew from the scrapings of the pseudo-membranous patches, together with considerable numbers of scattering forms. Among the latter were a few staphylococci pyogenes aureus.

CASE XXII.—*Clinical history:* Child, six days old. Developed severe umbilical phlegmon. Three days after this, diphtheria appeared, with severe local and general symptoms, with death on the following day. *Autopsy*, twenty-six hours: A dense, thick, firmly adherent membrane on the posterior surface of the epiglottis, extending down over the vocal cords and into the larger bronchi; commencing broncho-pneumonia, with much pulmonary congestion; parenchymatous degeneration in kidney. *Bacterial examination—Morphological:* In the false membrane enormous numbers of cocci, mostly single and in pairs, but many were in chains and irregular clusters. Many similar cocci were present in the lymph spaces of the submucosa and in the mucous glands of the trachea and larger bronchi. *Cultures: Streptococci* in large numbers. Staphylococcus pyogenes aureus. A few colonies of large bacilli resembling bacillus subtilis. Cultures from the tissues about the umbilical phlegmon revealed large numbers of streptococci and staphylococcus pyogenes aureus.

CASE XXIII.—*Clinical history:* Female, six years. Taken sick on Monday, with croupy cough and dyspnœa. No pseudo-membrane was visible. There was no evidence of systemic infection, but the dyspnœa increasing, tracheotomy was advised and refused by the parents. On Monday the child died. *Autopsy:* No pseudo-membrane in the pharynx. A dense, voluminous pseudo-membrane extended from the base of the epiglottis to the bifurcation of the trachea; this was firm and closely adherent above, leaving raw, rough surfaces when pulled away. Microscopical examination showed that, especially above, the pseudo-membrane had involved the external layers of mucous membrane *Bacterial examination—Morphological:* Few scattering bacteria were present in the pseudo-membrane, mostly on the surface; these were mostly larger and smaller short, stout bacilli. *Cultures:* No streptococci and no staphylococci were found. There were two forms of bacilli in considerable abundance, one a form common in the healthy mouth, and the other a short, plump, round-end bacillus. There were three or four scattering forms, mostly bacilli.

CASE XXIV.—*Clinical history:* Female, two and a half years. Croupy cough, dyspnœa, no membrane in pharynx, but strips of membrane coughed up. Constitutional symptoms became graver; tracheotomy; symptoms of broncho-pneumonia; death on the sixteenth day. *Autopsy*, twenty-four hours: Voluminous membrane in larynx, and trachea softened and loose below; no membrane in pharynx. Commencing broncho-pneumonia. Kidney swollen, cortex white and thick. Liver and spleen normal in appearance. *Bacterial examination—Morphological:* Pseudo-membrane thickly permeated by cocci, diplococci, and streptococci, with a few other scattering forms. Streptococci in considerable numbers in the lymph spaces of the submucosa. A few in the ducts and acini of the tracheal mucous glands and in the tracheal lymph nodes; large numbers of streptococci in the tissues about the tracheotomy wound; no bacteria of any form found in the kidney, liver, or spleen. *Cultures: Streptococci* in enormous numbers in the membrane with few scattering forms. Pure cultures in large numbers of streptococci in spleen, lungs, and liver. (Kidney cultures lost by accident.)

Before proceeding to study the characters and life-history of the streptococcus, to the importance of which our studies thus far seem to point,

we will group the cases of diphtheria examined and briefly summarize the results.

Sixteen of the 24 cases of diphtheria which we have studied, both morphologically and by the culture methods, Nos. I. to XVI., inclusive, occurred in a large foundling asylum with many inmates, in which the mortality each year, especially in winter, is large—particularly so during the prevalence of measles and scarlatina; 5 of these cases, Nos. XVII. to XXI., were from another large children's asylum in another part of the city, in which, also, during the winter season, the mortality from diphtheria is usually large; 1 case, No. XXII., was from a smaller children's asylum, in still another part of the city, in which diphtheria is of occasional occurrence; 2 cases, Nos. XXIII. and XXIV., were from private practice in an adjoining town. Nine of the cases were simple diphtheria, uncomplicated by any notable previous disorder; in 4 the local diphtheritic inflammation was preceded by a phlegmonous, suppurative, or erysipelatous inflammation in distant parts of the body; in 3 the diphtheritic inflammation was immediately preceded by well-marked scarlatina; in 7, by measles; in 1 by whooping-cough.

The only species of bacteria present in nearly all the cases (in all but two) in the pseudo-membrane was the *streptococcus*. This species was present in most of the cases in enormous numbers as shown by the cultures. In one case in which actual counts were made, it was found that from the invisible amount of material which clung to the tip of a fine sterilized platinum needle plunged into the softening pseudo-membrane from a bronchus, 124,300 colonies developed on the plates.

Cultures from the kidney, spleen, and liver were unfortunately not made except in some of the later cases; the results of these were as follows:[1] Kidney cultures in 3 cases; streptococci in pure culture in moderate numbers in 2 of them. Spleen cultures in 3 cases; streptococci in moderate or large numbers in 2 of them. Liver cultures in 3 cases; streptococci in moderate numbers in 1 of them. The streptococci were the only organisms found in these visceral cultures except in 1 case in which a few staphylococci aureus developed from the liver.

The streptococcus was in all of the cases not only by far the most abundant form of bacteria in the pseudo-membrane, as judged both by cultures and microscopical examination of stained specimens, but it was the only form which appeared to penetrate the underlying tissues. While the streptococcus was most abundant in the pseudo-membrane and in the underlying necrotic tissue—and in these situations it was often present in enormous numbers—it was also found, usually scattered singly or in large and smaller masses, in the ducts of the mucous glands and in the

[1] The bacteria found in the lungs in these cases will be described later in connection with studies not yet completed by Dr. W. P. Northrup and the writer on the etiology of broncho-pneumonia of children.

lymph spaces of the submucosa. It was rarely found in the tracheal and bronchial lymph glands, and then only in the form of scattered single cocci or short chains. In the kidney, liver, and spleen I have never found the bacteria by staining even in those cases in which the culture method showed that it was present in small numbers. As judged by the culture method, the number of streptococci which these viscera contain is in nowise to be compared to the numbers present in the pseudo-membrane and underlying tissue. In the tonsil crypts, on the other hand, the number of streptococci found, both by the staining and by the culture methods, was often very large indeed.

The second most common form of bacteria in these cases was the staphylococcus pyogenes aureus or albus. While this species appears in cultures from the membranes in a large proportion of the cases, neither in numbers nor situation does it appear to bear any relationship to the extent or character of the pseudo-membrane. It was usually quite as abundant in the mouth or in the bronchial secretions as on the pseudo-membranes. It was more apt to be abundant when the membranes were softening, and in general its abundance seemed to bear a much closer relationship to the amount of catarrhal than of pseudo-membranous inflammation.

So far as the other species of bacteria are concerned, which were cultivated from these cases of diphtheria, some twenty different species were isolated, but they were either forms which control-examinations showed to be frequently present in the mouths or air-passages of healthy children, or were scattering forms occurring in greater or less numbers but with no uniformity.[1] The bacillus of Loeffler was not found in any of the cases.

In judging of the probable importance of the staphylococcus pyogenes aureus and albus which was so frequently present, although often in relatively small numbers, in our cases of diphtheria, it should be borne in mind that a long line of researches by a variety of observers has shown by actual cultivation that the staphylococcus pyogenes is very frequently to be found floating with the dust in the wards and operating-rooms of hospitals, and in many other places which are either overcrowded or filthy. It has, furthermore, been shown by repeated culture experiments that the staphylococcus pyogenes is a not infrequent inhabitant of the mouth and nose in health and disease. It has been found by Dr. T. M. Cheeseman in the dust of the operating-room in one of the large hospitals

[1] Two of the more common species occurring in the mouths of healthy children develop on agar plates in forms very closely resembling those of the streptococcus colonies in their early growth. These are, however, both short, stout bacilli, but the fact that when they first commence to grow they form delicate translucent dew-drop-like colonies quite similar to the streptococcus colonies might lead the inexperienced observer into error. In one of these forms, moreover, the short bacilli are apt to occur in chains which form a looped border to the colonies, closely simulating the beaded chains which fringe the edges of the streptococcus colonies in some stages of their growth on the agar plates. These I have called the "chain bacilli" of the mouth.

of this city. I have repeatedly found it floating in the dust of the rooms from which many of the cases above recorded of diphtheria were taken, and it has been found in some of the other rooms in the same hospital. I have cultivated it from the mouths of several children in the asylum from which most of our cases of diphtheria came who were apparently healthy, from the saliva in one case of whooping-cough, and in several cases of measles. I have found it in the mouths and throats of children and adults both well and ill in private houses and in our large city dispensaries. Finally, as stated above, the staphylococcus appears to bear no relationship whatsoever either in situation or numbers to the pseudo-membrane in the cases of diphtheria in which it was present. It would thus appear that from what we know of the biology and distribution of the staphylococcus, its occurrence in and about the membranes in so many of our cases of diphtheria cannot be regarded as of any especial significance so far as the etiology of the disease is concerned. It may act as a complicating factor in the disease—indeed, its well-known pathogenic properties would lead us to conjecture that it does—but this is at present the utmost significance which the facts will warrant us in attributing to it.

We are thus narrowed down in the analysis of the results of these examinations to the only species of bacteria which was present in nearly all of the twenty-four cases of diphtheria studied, namely, the streptococcus. To this species, then, we must now turn our attention.

The morphological and biological characters of the streptococcus found in these cases were fully worked out in each, and they were found exactly to coincide. The following characters, therefore, describe the species common to all the cases of diphtheria studied, and this species may be called, at least provisionally, *streptococcus diphtheriæ*.

Streptococcus Diphtheriæ.

Morphology.—Sphero-bacteria, or cocci, varying in diameter from 0.75 μ to 1.2 μ, average about 1 μ. When growing freely on quite moist surfaces, or in fluid culture-media, such as beef-tea, they are apt to form longer or shorter chains. There is a marked disposition in the cocci which form the chains to be closely united in pairs, and thus appear as diplococci when the chains are forcibly torn apart (see Fig. 1, Plate II.). As the cultures grow older individual cocci in the chains are apt to grow larger and more irregular in shape, apparently through involution changes. In rapidly growing cultures the individuals composing the chains appear not infrequently a little elongated in the stage preceding division, and a little flattened transversely to the axis of the chain when the division is just completed.

Biology.—These bacteria do not fluidify gelatine. The colonies are white to the naked eye when they have reached a considerable size, and under the microscope have a faint yellowish or brownish shimmer. They

are immobile; their growth is in general slow, but is hastened at the temperature of 37° C. (98.6° F.). No spores are formed. Growth occurs both at the surface and in the depth of the culture-media.

Gelatine plates. On the second day, minute, sharp-edged, coarsely granular dark colonies appear, and become very gradually larger. As they grow older the colonies in the depth of the gelatine frequently become rough and irregular at the edges. They spread slightly on the surface of the gelatine, forming rough, often looped-edged, very slightly elevated disks.

Agar plates (surface plants). At 37° C., after twenty-four hours, minute, translucent, colorless, dew-like colonies appear, which, as seen under the microscope, have even or irregular edges, a moderately finely granular surface, and a faint yellowish shimmer. As the colonies become older (two to five days) their surfaces, particularly if the agar becomes a little dry, are apt to appear more coarsely granular. The outline of the slowly spreading colonies, as the growth gets older, is apt to become more or less irregular, from the projection of loops and strings of cocci, which, if the culture surface remains moist, may form a slowly spreading delicate pellicle of variously curved and contorted chains (see Fig. 2, Plate II.). Not infrequently the older colonies are more or less zonulated from successive rings of peripheral growth.

Agar-glycerine plates (glycerine 5 to 10 per cent.). The growth is essentially the same as in simple agar.

Blood-serum plates. The growth is much the same as on agar.

In *Beef-tea*, at 37° C., the growth is moderately vigorous, forming in twenty-four to forty-eight hours a considerable number of delicate white flocculi, which gradually collect along the sides or at the bottom of the tube. Growth at air temperature similar but slower.

In *Gelatine Tubes* the growth appears, at the temperature of the room, in from twenty-four to forty-eight hours, as a delicate, white, exceedingly finely beaded streak along the puncture-line, and is scarcely visible on the surface. After several days the central portion of the puncture streak becomes dense and whitish, and is beset on all sides with more or less discrete, whitish, spheroidal colonies, which may become as large as one millimetre in diameter. At this time the surface growth may be scarcely perceptible, or may have spread a little as a delicate translucent pellicle. The gelatine is never fluidified.

In *Agar Tubes* the growth along the puncture-line is apt to be more even and continuous than in gelatine, and is never extensive, while on the surface it appears as a faint translucent sinuous-edged pellicle, rarely spreading more than 1 to 3 millimetres from the point of inoculation.

In *Blood-serum* tubes the growth is essentially the same as on agar, but a little more vigorous.

On *Potato* there is no visible growth, but there appears to be sometimes a very slight proliferation near the line of inoculation.

The streptococcus diphtheriæ is readily stained by the common aniline dyes, and both from the cultures and in the tissues retains the color when treated by Gram's method.

PATHOGENESIS.—The experiments upon animals were made with cultures from ten of the cases of diphtheria—Nos. III., VI., VIII., XIII., XV., XVI., XVII., XVIII., XIX., XXIV. These were cases selected at random from the different hospitals or houses from which most of the material was derived. Vigorously growing beef-tea cultures were, for the most part, employed from two to five days old. Such cultures are slightly turbid when shaken to break up the flocculent growth, and quantities varying from five drops to two cubic centimetres were used for each inoculation. The inoculations were performed with the usual precautions against accidental contamination. The very convenient Sternberg sterilizing injecting syringe was for the most part used. It does not seem to me necessary to give in detail a history of each of the inoculated animals, since the general results, so far as our subject is concerned, are the ends sought for.

Altogether, 80 animals—rabbits, hens, and pigeons—were inoculated with the streptococcus. The following is a summary of the results:

Hens. In hens (8 animals), the subcutaneous inoculation and the inoculation into the scraped or punctured mucous membrane of the mouth or trachea were entirely without result, save for a temporary redness of the mucous membranes following the local mechanical injury. Subcutaneous inoculation gave no results.

Rabbits. Intravenous injections (9 animals). The injection of from ½ to 1½ c. c. of beef-tea culture was made, with no marked effect in any case.

Subcutaneous and *intramuscular* injection (10 animals). These inoculations were made in the pectoral or thigh muscles, or beneath the skin in these regions. In 7 of the cases, there was a considerable formation of pus at the seat of inoculation, usually in the form of a small, circumscribed abscess. The streptococci were readily cultivated from these abscesses, unless they had become old and the pus quite dry and grumous, when they were found in very small numbers, or not at all. In 3 out of the 10 cases, the result was negative.

Injection into the subcutaneous tissue of the ear of the rabbit (24 animals). In 1, there was no effect whatsoever. In 4, there was a slight, diffuse redness about the seat of inoculation, passing away on the second or third day. In 5, there was slight local redness, on the second day passing off, and the formation of a small subcutaneous abscess from the third to the eighth day. In 2, there was moderate local redness, followed by death, under symptoms of progressive weakness, on the second and

fourth days. In 12 cases, there was well-marked erysipelatous redness of the ear, extending, sometimes, for an inch about the seat of inoculation, but most often involving the whole ear, and extending down into the head. The redness and circumscribed thickening of the ear in these cases were usually sharply defined and slowly progressive, as in typical erysipelas. The inflammation was established on the second or third day, accompanied by a rise of temperature of from 1° to 3° C. (32.8° to 37.4° F.). Resolution usually began on the fourth day. In 7 cases, resolution was complete; in 3, a small abscess formed at the seat of inoculation; in 2 cases death occurred, and in the second on the fourth day. No internal lesions were ever found in these cases of erysipelatous inflammation of the ear, nor was there, except in the fatal cases, much evidence of systemic disturbance. Streptococci were cultivated from the fluid exudation at the seat of the inoculation. The erysipelatous inflammation was more marked and intense if the tissue of the ear was pierced or undermined in various directions by a sharp, sterilized needle before the bacteria were introduced, by the method of Biondi (12).

Injection into the anterior chamber of the eye (6 animals). A moderate conjunctivitis occurred in 2 cases, with opacity of the cornea about the puncture, and a moderate accumulation of fibrin and pus in the anterior chamber. All of these evidences of inflammation began to subside on the fourth day, and within two weeks the exudation had largely disappeared. In 4 of the cases, an intense panophthalmitis occurred, with destruction of the eye.

Inoculations into the mucous membranes (6 animals). These were accomplished, in 3 animals, in the trachea by tracheotomy, the mucous membrane being scraped, and the culture rubbed into the abraded surface; in 2 animals, in the mucous membrane beneath the tongue; and in 1 animal, in the vaginal mucous membrane. In none of the animals was there any marked reaction, either local or general.

Pigeons. The inoculations in these animals (17 in number) were made on the mucous membrane of the mouth beneath the tongue, or in the larynx[1] and upper end of the trachea. The operation consisted either in scarifying the mucous membrane with a sterilized scalpel or needle, and then firmly rubbing in the culture; or in making a submucous injection of a few drops of the same.

The general effects may be briefly summarized: In 2 animals, there was no effect whatsoever, either local or general. In nearly all of the cases there were moderate redness and swelling about the seat of inoculation, on the second day. This, in 4 cases, was accompanied by a yellowish-white pellicle from 0.5 to 1 mm. in thickness, which did not spread, and consisted of necrotic mucous membrane containing micrococci similar

[1] Owing to the conformation of the pigeon's throat, and the great mobility of the larynx, the upper end of the trachea is readily accessible through the mouth.

to those injected. In 3 of the cases of tracheal inoculation, in addition to dusky redness and swelling of the mucous membrane, there was a white, dense, stringy, moderately firmly coherent pellicle, over the affected surfaces. This in no case tended to spread, and consisted largely of mucus. About one-half of the animals died, from the second to the sixth day, without dyspnœa or symptoms other than those of progressive feebleness. Of the animals which showed a moderate degree of local and inflammatory reaction, 2 were sick for three or four days, and then completely recovered, while 3 were apparently not sick at all.

We thus find that the reaction of animals to the inoculation of the streptococcus diphtheriæ varies a good deal with the species of animal experimented upon. While chickens are, in general, invulnerable, rabbits and pigeons show a marked, though varying, degree of susceptibility. In general, it may be said that young animals are markedly more susceptible to its action than old ones. The general effect is to produce either suppuration or erysipelas, or local necrosis or septicæmia, without marked lesions. Sometimes, one or more of these general effects are associated as the result of the same inoculation.

It is thus evident that the streptococcus diphtheriæ, as cultivated from our group of cases of diphtheria, though not inducing in the animal experiments which I have done a disease which can be regarded as diphtheria, is yet, in some animals, markedly pathogenic.

Control Cases.

For the purpose of control, I have made cultures from the scrapings of the throat and especially the tonsils of twenty-five healthy children, living in private houses or in a new and well-aired maternity hospital, and in these I have not found a single colony of the streptococcus.

I further made cultures from the throat and tonsil scrapings of six cases in private practice during illness—two of measles, two of scarlatina, one of follicular tonsillitis, and one of quinsy. In two out of these six cases, large numbers of streptococci identical in every respect with those isolated from our cases of diphtheria were found in enormous numbers. Their effects were tested on animals. The other four cases gave no streptococci. The two cases in which the streptococci were found were the cases of scarlatina occurring in the same house. This is especially noteworthy, as, within one week of the time of the examination, diphtheria of a severe form developed in both of the children; going on, however, to final recovery, so that no further examinations were made. Thus, out of thirty-one control cases, the streptococcus was found in only two, and these were cases of scarlatina in private practice which shortly developed diphtheria.

Conclusions from Experiments.

We are now ready to see what conclusions can be justly drawn from these researches as to the etiology of the cases of diphtheria which we have studied in detail. It is a well-known principle founded upon the modern methods of research, that in the investigation of the etiology of any acute infectious disease we should seek to ascertain first whether there are special forms of bacteria present in the lesions with such uniformity as to justify the conjecture that they may stand in an etiological relation to it. The answer to this question we have found in our cases of diphtheria in the nearly uniform presence in significant situations of a streptococcus. Second, we should seek to isolate in pure forms and to learn by cultivation under varying conditions the life-history of the suspected organism. This has been done in detail in every one of the cases of diphtheria examined. Third, we should, by inoculations of the pure cultures of the suspected organisms into animals, learn whether they are capable of producing disease or not, and especially should we endeavor to induce a form of disease similar to that from whose lesions the organisms were derived. We have found in pursuing the third line of inquiry that, while the suspected bacteria were markedly pathogenic, we have not been able to induce, by their inoculation in animals, a disease which can fairly be regarded as diphtheria.

Let us now see whether the failure to induce diphtheria in animals really means that the streptococcus which we find under such suspicious conditions in the human disease is really of no significance as an etiological factor. In the first place, it is very doubtful whether any of our domestic animals, or any of those which are commonly employed for experimental purposes, are ever spontaneously subject to diphtheria as we know it in man. There is, indeed, a disease common enough in calves, in fowls, in pigeons, and in rabbits, in some parts of the world, which is called diphtheria, and which is associated with the development of a false membrane on the mucous surfaces; but, so far as these have been investigated by the modern methods, it appears that in none of these are the same bacteria found as are present in the human disease; and, moreover, the species of bacteria which are found, and which are presumedly the cause of the disease in animals, differ entirely in the different animals.

Moreover, although there is a considerable number of fairly well authenticated instances in which the animal diseases from one species or another have been communicated to man with the development of the general symptoms of diphtheria, and a fairly typical local lesion, it may well be questioned whether there is anything more than a general analogy between this disease and genuine human diphtheria; for, after all, as we have seen above, it is the etiological factor which we must ultimately depend upon for the identification of the disease. On the other hand,

although many hundreds, perhaps thousands, of attempts have been made to reproduce the human disease in animals by inoculation either of the fresh membranes or cultures, more or less pure, of various microörganisms isolated from them, there does not appear to have been a single case in which an effect was induced which could fairly be absolutely identified with the human disease. We are thus very unwillingly led to face the possibility that animals are not subject artificially, as they appear not to be naturally, to diphtheria as we know it in man.

Now in a disease which is accompanied with a definite and constant lesion, such, for example, as tuberculosis, the problem of the significance of the results of inoculations in animals is much more simple. But in diphtheria, as in typhoid fever and some other diseases whose local lesions are variable and may be absent altogether, we must recognize the possibility that after all we may be forced, by the nature of the case, to do without the light which successful animal inoculations might throw upon the problem of their etiology. In other words, there is a growing probability that there may be acute infectious diseases to which animals are neither naturally nor experimentally subject. If such should prove to be the case, we may find ourselves perforce obliged to rely upon the evidence which the constant occurrence of a definite species of bacteria in a given disease in a large number of cases can furnish, and upon the side light which can be thrown upon the problem by a variety of collateral experimental and clinical observations.

I would not be understood to say definitely that diphtheria, or any other acute infectious disease, cannot be produced artificially in animals. I wish only to call attention to the fact that, so far as the evidence goes, genuine diphtheria as we know it in man has not, in my opinion, up to the present time been thus induced.

Now, we have found in all but two of the twenty-four cases of diphtheria examined a distinctly pathogenic species of bacteria with great uniformity, a species of bacteria which does not, indeed, induce diphtheria in the animals experimented upon, but does induce allied forms of inflammation. Moreover, we have not found in any of the cases the bacilli of Klebs–Loeffler, nor any other to which any significance can justly be attributed.

It may, of course, be said that these streptococci may not actually be the cause of the disease, though so constantly present; that the disease may be induced by some other unknown agency, some germ, perhaps, which does not grow under the artificial conditions which we supply; and that it is simply the furnishing by the lesion of a favorable growth-medium for the streptococcus which may explain its common occurrence. That all this may be true must be admitted; that it is so it needs facts as yet undiscovered, and to which there is no clew, to make probable. On the whole, it seems to me that, considering the insusceptibility of

animals to diphtheria as we know it in man, the result of all of these series of experiments taken together abundantly justifies us in the assumption that in a certain class of cases, at least, diphtheria is caused by a streptococcus having the characters set forth above.

Remarks on Various Features of the Cases of Diphtheria.

As to an explanation of the apparent absence of the streptococcus in two of the cases studied—Nos. XVI. and XXIII.—I have none to offer. It is, of course, possible that they may have been at some time present and died out, but I have found them in the greatest abundance in cases of equal or greater duration of the disease. It is quite possible that by some technical inadvertence I may have failed to find them, although these cases were studied with the same detail and care as were the others. It is, furthermore, possible that, although called diphtheria, in accordance with the views laid down early in this paper, these apparently exceptional cases may have been examples of simple pseudo-membranous laryngitis or croup. Indeed, Case No. XXIII. was diagnosticated as "croup" by the attending physician, with the distinct specification that there was no clinical evidence of "diphtheria" in the case. The child died from strangulation, the parents having refused operation. But within a few days, in the same house and family, another child was similarly attacked, but with symptoms of marked constitutional disturbance, and shortly died. In this second child (Case XXIV.) the streptococci were found not only in the membrane, but in small numbers in the viscera.

It will be remarked, in the cases of diphtheria in which cultures were made, not only from the local lesions, but from the kidney, liver, and spleen, that, while the streptococci were usually present in the pseudo-membranes and in the tissues just beneath it in very large numbers, they were, when found in the viscera at all, only present in very small numbers indeed. They were also usually absent, or present only in very small numbers, in the tracheal and bronchial glands, although these are apt to be red and swollen and their lymph sinuses to contain fibrin. This condition of affairs, taken together with the early and severe symptoms of systemic infection and the lesions of parenchymatous degeneration of the viscera, especially the kidneys, which are apt to occur in diphtheria, would seem to indicate that the systemic changes are probably due to the absorption of some soluble poison (ptomaine) produced where the streptococci are most actively growing, namely, at the seat of local lesion, or possibly, as will be seen later, in the lungs.

Metastatic septic visceral inflammations were present in none of the cases examined, and are, as is well known, not very common in diphtheria.

In nearly all of the cases of diphtheria in our series the pseudo-

membrane was moderately developed or very abundant. In two, however, complicating measles, Nos. XX. and XXI., death occurred so early that in one case there was no pseudo-membrane found, and in the other but two small, isolated fragments.[1]

The detailed study of these early fatal cases, both morphologically and by the culture methods which served to identify the species of bacteria present, throw, as it seems to me, a good deal of light upon the manner of local invasion of the disease.

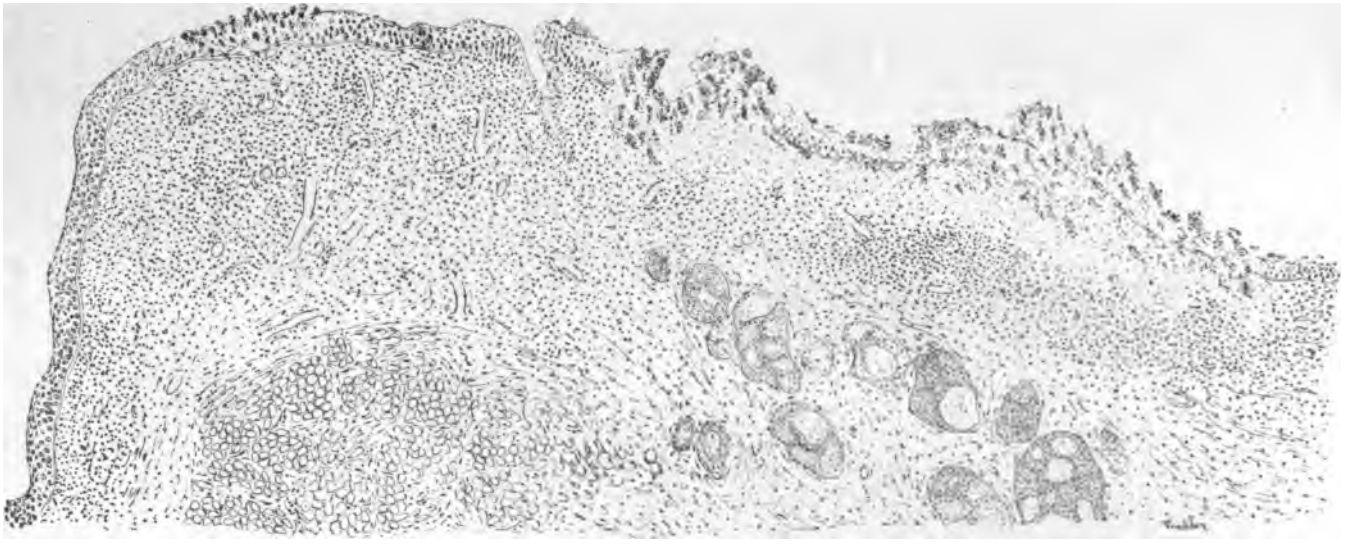
In the case (No. XX.) in which no pseudo-membrane had formed there were the lesions of an intense catarrhal pharyngitis and laryngitis. The bloodvessels were congested, there was an increased amount of mucus on the surface, and the epithelium was in places loosened and proliferating. It was found, both by the microscopical examination and by cultures, that streptococci were scattered sparsely over these inflamed surfaces. In two situations, however, the streptococci were found in considerable masses, namely, in one of the tonsillar crypts and in the ventricle of the larynx. In the tonsil crypts, distinct though small areas of necrosis of the epithelium were found in the immediate vicinity of the bacterial masses. In the ventricle of the larynx no evidence of necrosis was found, but the epithelium was in places detached, and streptococci were found beneath and between them, and in small numbers in the adjacent submucosa.

In Case XXI., on either side of the base of the epiglottis in the pharynx were small white patches, which had the gross appearance of pseudo-membrane. The microscopical examination showed that in these situations there was a considerable heaping up and detachment of the epithelium, with partial necrosis of the cells and of the submucosa beneath, while streptococci and single cocci were scattered and massed among the cells and in the necrotic tissue beneath them. In the same case the small pseudo-membranous patches just below the rima glottidis showed necrosis and partial detachment of the epithelium, which, mingled with granular material and large number of cocci, singly, in pairs, and in chains, formed a well-defined, irregular pellicle. The submucous tissue beneath this pellicle was superficially infiltrated with similar bacteria, and was necrotic (see Plate IV.).

The abdominal viscera in these two cases were not sent to me, so that no bacterial examination was made of them, and so slight were the local lesions that it would seem at first as if we were hardly justified in assuming that death was due to diphtheria unless there had been a systemic infection from some source other than the pharynx and larynx. In Case XX., however, although there was but a very slight amount of broncho-pneumonia, the air spaces of the lungs contained enormous numbers of cocci and streptococci. It is quite possible—and Dr. Northrup

[1] These two cases belonged in a group of five cases of measles with diphtheria sick at about the same time and in the same place. In two of these voluminous pseudo-membranes formed, the duration of the diphtheritic symptoms being longer than in the non-membranous cases, in which death occurred almost at the outset of the disease. These facts, together with the results of the bacterial examinations, seem to justify the diagnosis of diphtheria.

PLATE IV.



Longitudinal section of larynx, just below *rima glottidis*, in diphtheria (Case XXI.) showing the commencement of the formation of a pseudo-membrane, and the infiltration of the involved tissues with streptococcus masses.

and I have already made numerous observations which speak strongly in favor of the view—that in cases like these the early death in diphtheria may be due to the absorption from the lungs of a bacterial poison before either a pneumonia has developed or a pseudo-membrane formed.

I have been much impressed in the course of these studies with the disproportion which often exists between the amount and the extent of the pseudo-membrane, particularly of the larynx and trachea, and the actual involvement of the underlying mucous membrane. There are, I think, always to be found some regions where the pseudo-membrane is closely attached to and is formed in part by the epithelium and the superficial layers of the submucosa; but in almost all cases the pseudo-membrane spreads away over areas of mucosa where the epithelium is quite intact, and its underlying tissue, save, perhaps, for congestion and œdema, is perfectly unchanged. Very often I have found a closely adherent pseudo-membrane extending from the tip of the epiglottis down over the vocal cords, or simply about the cords themselves, which was made up in large part of the superficial layers of the mucosa; while below the vocal cords a more voluminous and largely fibrinous false membrane extending through the trachea and even into the small bronchi left the mucous membranes of these tubes virtually intact. It would seem that a comparatively small area of actual involvement of the mucous membrane can, in the larynx and trachea at least, furnish the material for a fibrinous pellicle of great extent. In the pharynx, however, the extent of the pseudo-membrane is much more nearly coincident, in my experience, with the involvement of the mucous membrane, than in the larynx and trachea. This condition may serve, partly at least, to account for the disproportion which is so often observed between the apparent extent of the local lesions and the systemic infection, and may in part explain the absence of the symptoms of general infection in some cases which, on clinical grounds, has appeared for so long to justify the separation of croupous laryngitis from diphtheria.

Examinations of Children Exposed to Diphtheria.

As we are not able to confirm by the successful induction of typical diphtheria in animals, the conclusions to which we are led by our morphological and biological studies on diphtheria, it seems highly important to draw all the data possible from a careful study of the conditions under which children are placed in hospitals or elsewhere where diphtheria is prevalent among them, since valuable evidence, confirmatory or otherwise, might, it seems, be arrived at in this way. With this end in view, I have made a series of studies on children not the victims of the disease, but associated with those who were suffering from it in the hospital in which it was frequently occurring and from which most of our cases were taken.

In the first place, with the concurrence of Dr. Northrup, I examined, by the culture methods, scrapings from the tonsils of 25 children both well and ill. In 17 of these cases no streptococci were found. In 8 of the cases the streptococci were found in small numbers. All of these 8 positive cases had been for some time in the hospital and in rooms in which diphtheria was liable to occur. One of these cases in which the streptococcus was found had been in the room with the mother, who was the victim of puerperal fever. This child died soon afterward from a severe umbilical phlegmon, and from the inflamed tissues large numbers of similar streptococci were cultivated. Two of the cases were suffering from measles. They were quarantined at about the time the examinations were made, and soon after developed diphtheria from which they died. Another of the positive cases, convalescing from broncho-pneumonia was confined in a small room with twelve inmates. Two other of the positive cases had been for a considerable period in a large room with many inmates, and *from the dust of this room the streptococcus was cultivated.*

In a second series of examinations cultures were made from the tonsils of children which had been for varying periods in the same hospital, and had died of various diseases not diphtheria. There were in this class fifteen cases; twelve of these were cases of marasmus with more or less broncho-pneumonia or entero-colitis; one was a case of pertussis with extensive interstitial emphysema; one, which had been for a long time in the hospital, died with symptoms of suffocation, the autopsy revealing only œdema of the lungs; the other was a case of erysipelas following vaccination and extending over the entire arm and chest. Of the 12 marasmus cases, 7 showed no streptococci; in 4 the streptococci were found in small numbers, but only in deep scrapings from the tonsillar crypts and folds. These 4 cases in which the streptococcus was found had all been for considerable periods in the hospital; they were all confined in rooms in which at the time diphtheria was occasionally originating. One of these had been for several days in a room with a number of eczema cases, another in the "eye-room" with a number of cases of purulent conjunctivitis. Of the 3 remaining cases, the one with pertussis showed no streptococci; the one with pulmonary œdema and the one with erysipelas showed a few. We thus see that from scrapings, mostly from the tonsillar crypts or the folds of mucous membrane about them in 15 children dead of various diseases not diphtheria, in a large children's asylum in which diphtheria was constantly occurring, the streptococcus was found in 6.

If now we summarize the results of the cultures of scrapings from both the dead and living children in this institution in which diphtheria was prevalent, we find that in 12 cases out of 40 small numbers of streptococci were present in and about the tonsils of children not at the time suffering from diphtheria. In 2 of these, fatal diphtheria shortly

developed. The comparison of this result with that of similar cultures from the tonsils and throats of well and sick children outside of this hospital and mostly in private practice given above is striking. For in these, from scrapings from 31 cases, the streptococcus was cultivated in only 2 which were suffering from scarlatina at the time, and which both developed diphtheria shortly after. The significance of these collateral observations is, I think, evident and needs no comment.

It should be said that the identity of the streptococci isolated from these control cases with those found in diphtheria was established, not only by their morphological and biological characters, but by the results of their inoculation in animals, the details of which it is not necessary to enter into here. The only difference which I observed between the streptococci from the control cases and those from diphtheritic membranes was that, in general, the former were, for the most part, a little slower in their growth, and their effects upon animals a little less pronounced. They produced, in a large proportion of the inoculations, either erysipelas, inflammation in the ear of rabbits, or small abscesses; but the abscesses were invariably small, and the inflammatory reaction short-lived.

The Relationship of the Streptococcus Diphtheriæ to other Species of Bacteria.

We have now arrived, by an independent series of observations on a group of cases of diphtheria, at a definite and precise notion of the species of bacteria which appears to stand in a causative relation to the disease. If we compare this species, which we have called the streptococcus diphtheriæ, with those species of bacteria already known and fully described, we find that, in its forms and in its modes of growth, as well as in its effects when injected beneath the skin or into the blood of animals, it appears to be identical with two well-known species, called streptococcus pyogenes and streptococcus erysipelatos. I have carried the cultures of streptococci from cases of diphtheria along side by side with cultures of streptococci made from various cases of simple acute erysipelas and simple phlegmonous inflammation, week after week; over and over again, I have stained, measured, and compared the growth from these three sets of sources. I have repeatedly inoculated duplicate sets of animals with the different cultures, and I have never found a single appreciable constant feature of difference between them. There are, it is true, often differences in rapidity and vigor of growth on artificial culture media, as well as differences of virulence, as the result of inoculation of animals, between cultures derived from the different sources. But these differences are no more marked between cultures from erysipelas, phlegmon, and diphtheria than those often observed between cultures derived from different cases of the same disease.

While it is not possible, with our present knowledge, to characterize

precisely the difference in action, upon the animal tissues, of the streptococcus pyogenes and the staphylococcus pyogenes, associated, as they so often are, in the phlegmonous inflammations; in general, the streptococcus appears to be more prone to cause the spreading, progressive form of inflammation, and to give rise to general infection, than is the staphylococcus, the more marked tendency of which is to give rise to localized suppurative inflammations, abscesses, etc. Both forms of bacteria may, indeed, induce suppuration and abscess, and both may cause more or less widely spreading phlegmonous inflammation; but the special tendency of each appears to be that which I have stated.

It is worthy of note, in this connection, how often the streptococcus has been found associated, apparently as the etiological factor, with those inflammations, particularly of the serous membranes, in which the formation of fibrin is a most marked feature.

To enter here into the story of the discussions as to the identity of the streptococcus of erysipelas and the streptococcus pyogenes would lead us far afield, and bring us at last to no very definite result. The fact is, that nearly every practical worker in bacteriology, who concerns himself with these particular species, appears to find himself forced by his observations to the conclusion that, in their morphological and biological, and, for the most part, in their pathogenic, characters, so far as animals are concerned, the streptococcus pyogenes and streptococcus erysipelatos are practically identical. Nearly every one, however, hesitates to declare absolutely their identity, not on the ground of morphological or biological or experimental discrepancies, but rather because clinical experience and observation have for so long sanctioned the belief that erysipelas and simple phlegmonous inflammation are distinct and separate diseases. The close relationship existing between these three forms of inflammation has long been the subject of observation and speculation, and the probable unity of the causative bacterial agent has been set forth with much interesting detail and critical acumen by Baumgarten (14), as well as hinted at by others.

The Relationship of Diphtheria to Erysipelas and Phlegmonous Inflammation.

Whether the clinical differences between erysipelas and those forms of suppuration in which the streptococcus pyogenes occurs cannot be explained—perhaps, by the mode or seat of invasion, by variation in virulence or the resisting capacity in the individual, or in some other way—are questions which further observations must answer. That a simple erysipelatous and a phlegmonous or suppurative inflammation very frequently merge into one another is well enough known to all. That different effects should be produced by the same species of bacteria growing in the lymph spaces of the skin, or in the depth of other forms of tissue, or on the mucous membranes or wound surfaces, is by no means incom-

prehensible, and is not without abundant analogies in the life-history of other organisms. What the exact bearing of these differing conditions under which the bacteria grow, and what their relations may be to the symptoms and lesions, are matters for further investigation. But it is, after all, the pseudo-membrane—a structure either fibrinous or necrotic, or both, in its nature, and the seat of its most frequent occurrence, namely, the upper air-passages—which especially characterizes diphtheria in its common phases. Now, given a bacterium, like the streptococcus, capable of inducing a marked irritation and necrosis in the tissues, and set it growing in such enormous numbers as are seen in many cases of diphtheria on a mucous membrane, or on the granulation tissue of a healing wound, where the smaller bloodvessels are abundant and close at hand, and the conditions are abundantly fulfilled for the induction of emigration of leucocytes, formation of fibrin, and localized necrosis of the involved tissues—the conditions, in other words, for the development of a necro-inflammatory pellicle over the affected surfaces. At any rate, the position into which we are led by these studies is simply this: There seems to be experimental ground for the belief that some forms, at least, of diphtheria and erysipelas, and some forms of phlegmonous inflammation, are phases of the inflammatory process having one, at least, of their prominent etiological factors in common, namely, the inciting species of bacteria.

It should be remembered that to admit this is by no means to identify these diseases: no important clinical tradition is thereby overthrown. The different phases of inflammation become, it is true, by this conception of them more intimately related to one another. But, on the other hand, those factors which determine that infection with the one form of germ shall induce now a diphtheritic, now a phlegmonous, and now an erysipelatous inflammation, become, from this point of view, of even greater importance than before.

However iconoclastic this view may seem to those who have been accustomed to regard these phases of inflammation as entirely distinct, there seems to be a large amount of evidence other than that derived from experiment which favors the view to which these studies, somewhat unwillingly it must be confessed, have led the writer. In the first place, every practising physician knows how frequently suppuration occurs as a local complication of the diphtheritic process. The occasional simultaneous occurrence of severe umbilical phlegmon with pharyngeal and laryngeal diphtheria in the same child is not without significance in this connection, especially in view of the identification, in two of the cases noted above, of the same species of streptococcus, both in the suppurating tissues about the umbilicus and in the pseudo-membrane in the air-passages.

The occurrence of a diphtheritic inflammation of the mucous mem-

brane of the uterus, which occasionally forms one of the manifestations of puerperal fever, in connection with which the streptococcus pyogenes has so often been demonstrated, is also a significant fact.

The reputation of diphtheria as a sewage and filth disease is much more comprehensible than before when once we recognize the close relationship which seems to exist between at least some of its forms and the suppurative diseases. If the bacteria which act as the inciting cause of diphtheritic inflammation be forms peculiar to this disease alone, it is rather difficult to account for the occurrence of sporadic cases of diphtheria in which there is no evidence of direct communication of the disease from one individual to another. But if we admit the causal relationship between the streptococcus of suppuration or erysipelas, and some cases of diphtheria, nothing could be more evident, because the discharges from all sorts of suppurating wounds and surfaces form a very common and important bacterial element, not only in sewage, but in the air of our crowded dwellings and hospitals. But we need not go with more detail into these general considerations, which the experiences of every practising physician will enable him richly to supplement.

It should, however, be always borne in mind that in most of the acute infectious diseases as we see them in man, while the bacteria serve as the inciting cause, without which the particular form of disease could not, as we believe, exist, there are other and most important contributory factors which can by no means be ignored, and which are daily assuming a greater and greater importance. These contributory factors to the occurrence of an acute infectious disease are often those to which the attention of the practitioner has been more especially directed in his observations on these diseases, and, consequently, are often regarded by him as the etiological factors themselves. Thus it was that at the announcement by Koch, in 1882, that the bacillus tuberculosis was the etiological factor in tubercular inflammation, many whose attention had been prominently directed toward the important influence of hereditary predisposition, were disposed to doubt, on this ground alone, the importance of the bacillus in causing the disease. A similar condition of affairs has been revealed in acute lobar pneumonia. The importance of exposure to cold and wet, injury, etc., in inducing the disease is just as evident now as ever it was, only we are now aware that the exposure, etc., appear to be but predisposing factors, which explain why in one case an individual may harbor in his saliva or elsewhere the bacterium of pneumonia without experiencing the slightest evil, while in another, exposure or other deleterious agencies rendering the conditions favorable for the development of the germ, an acute lobar pneumonia may ensue.

The important relationship between measles and diphtheria, and between scarlatina and diphtheria, appears in the light of these studies *but* another illustration of the same conditions. The universally recog-

nized danger in both measles and scarlatina, namely, that diphtheria may occur as a complication, is amply illustrated by the history of epidemics of both measles and scarlatina in any of our large children's hospitals. That tissues which are ordinarily invulnerable to the growth and action of pathogenic bacteria may become very susceptible to their presence by a slight chemical change in the material present, is shown by the recent striking experiments of Bujwid (15), who found that the introduction of a small amount of solution of grape sugar into tissues which did not usually react at all in the presence of the staphylococcus pyogenes aureus would lead them to do so readily with the production of abscess. That some such slight change in the exudation of the mucous membrane of the air-passages as occurs in the catarrhal inflammation of scarlatina and measles might favor the growth of bacteria which had lain dormant and harmless in the tonsillar crypts or elsewhere in health, seems, in the light of these studies, a justifiable hypothesis. With the concurrence of Dr. Northrup, I have endeavored to ascertain whether any change in the reaction of the fluids of the mouth and throat occurred at the advent of measles and scarlatina which would render them more favorable for the growth of the streptococcus; but our studies in this direction have thus far led to no tangible results.

It should be distinctly understood that these studies on the etiology of diphtheria and the conclusions derived from them apply only to a certain set of cases. It by no means follows that because this particular group of cases of diphtheria is caused by a streptococcus, and seems to be intimately related in etiology to some other common phases of inflammation, that all cases of diphtheria are induced by the same organism or are related in the same way to erysipelas and phlegmon.

That other organisms than the streptococcus here described may induce that set of symptoms and lesions to which we give the name diphtheria, we must, of course, admit, and this the more readily on account of the great variability of both symptoms and lesions in even well-marked groups of cases. So far as Loeffler's experiments go, they tend to indicate that some other forms of bacteria really may play a part in the induction of certain forms of the disease. But it is not without significance that Loeffler also found a streptococcus very similar to that which I have found in our cases, not only in connection with his bacillus, but in several cases in which the bacillus was absent.

The statement of Loeffler, that the cases in which the streptococcus was present were apt to be those in which the pseudo-membrane was not greatly developed, and in which the necrotic character of the inflammation prevailed, certainly does not apply to the cases which I have studied; for in more than one-half of my cases the pseudo-membrane was both voluminous and extensive.

The fact that Klebs, Heubner and Bahrdt, Babes, Emmerich, Oertel

Baumgarten (16), and many others have been led on either morphological or biological grounds to attribute much significance to a streptococcus in causing diphtheria should be placed in evidence here.

If the streptococcus which we find so constantly in diphtheria be specifically identical with the streptococcus of phlegmonous inflammation and of erysipelas, the propriety of giving it a special name might well be called in question from the point of view of scientific nomenclature. But when we consider that bacteriology is still in its earliest infancy, and that both its classifications and nomenclature are only tentative and temporary, pending a greater accumulation of facts, such a special name without prejudice to future revision may serve for use until the whole bearings of the subject are better defined.

The Effects of Drying on the Streptococcus Diphtheriæ.

In view of the fact that diphtheria may be communicated by portions of the inflammatory exudate containing the streptococci, it seemed desirable to learn how long the germ could resist the deleterious effects of drying, such as would occur should portions of the exudate become distributed about rooms or upon fabrics.

The first set of experiments was made upon pure cultures of the streptococcus. Fresh and actively growing beef-tea cultures from three to five days old were prepared, and, after being thoroughly shaken so as to distribute the bacteria uniformly through them, sterilized bits of silk thread, pieces of flannel, etc., were soaked in them for a few moments and then dried in sterilized flasks and tubes. These fragments of thread and fabric were now planted from time to time in order to see whether the bacteria had been killed or not by the drying. This set of experiments was done with cultures from five of the diphtheria cases.

I have not sought to obtain numerical results in these experiments because I soon found that the streptococci from the different cases varied a good deal in their capacity to remain alive in the dried condition. In general, it may be said that a considerable number of the germs are killed by drying, but in all of the experiments a very large number were not killed. Thus I have at intervals from eight to ten days replanted the dried germs, and, up to eighty-five days, the longest period to which I have carried the experiment, large numbers grew most vigorously from the dried cultures. I have inoculated rabbits with the offspring of these dried germs and found them extremely virulent, inducing marked and violent erysipelatous inflammation. I have found, however, that the streptococci from some of the cases, while resisting the drying very well, and retaining the power when replanted of growing vigorously, had, nevertheless, become much less resistant to the action of the germicidal fluids than they were before drying.

But, as it might be objected that the results of drying the pure cultures

would not necessarily be the same as the drying of the streptococci in the membranes themselves, as might happen in practice, I have dried fragments of the fresh membranes from a number of the cases on the smooth walls of tubes and flasks. These dried bits of membrane I have scraped off from time to time, and in every case up to fifty-four days, the longest period to which any experiment was carried, large numbers of streptococci grew vigorously, together with some other forms of bacteria.

Thus we find, as the result of these drying experiments, that the streptococcus diphtheriæ when dried on smooth surfaces or fabrics, either in the form of pure cultures or in the crude pseudo-membranes, may retain its vitality and virulence for long periods.

Effects of Freezing on the Streptococcus Diphtheriæ.

As the possibility has been suggested that the exposure of infected rooms and their contents to the cold in winter might accomplish the destruction of the contagion of diphtheria, I have exposed, to a temperature below freezing, the streptococci from several of the cases of diphtheria placed on fabrics. The germs in one of the sets of experiments were dry; in another were thoroughly wet. The exposure to a temperature of —10° C. (14° F.) for seventeen hours did not destroy the vitality of any considerable proportion of the germs.

Exposure of the pseudo-membrane from two of the cases of diphtheria, both in the wet and dry conditions, to the same temperature for the same period, showed that under these conditions also the germs may resist an extreme degree of cold.

The conditions under which steam sterilization is effective have been so frequently and so fully worked out that the effects of high temperature upon the streptococcus diphtheriæ were not studied.

The Effects of Germicides on the Streptococcus Diphtheriæ.

Sulphurous acid. Although the vapor of burning sulphur has been repeatedly shown to be of but very moderate value as a germicide, the fact that it is still so generally relied upon in this country, especially for the disinfection of rooms, has led me to carry out a series of experiments on its effects upon the streptococcus diphtheriæ.

In these experiments, I have used a glass bell-jar, of about one gallon capacity, into which the objects to be treated were lowered by means of fine platinum wires through an opening in the top. The articles were placed in little metallic baskets, which were carefully sterilized before each experiment. The sulphur was burned in a small dish at the bottom of the receiver, and the whole apparatus was tightly sealed, top and bottom, at each operation. The receiver, in all cases, remained closed for twenty-four hours, and, when opened, the contained air was invariably

still strongly charged with the vapor. It is evident that the conditions under which the experiments were done with this apparatus were much more favorable for the action of the sulphurous acid than in the disinfection as it is usually practised in larger chambers which are rarely, if ever, perfectly tight.

I first tried the power of the sulphur vapor to kill the streptococcus in pure cultures, in a perfectly dry condition, in the interstices of threads and fabrics. Cultures from several of the cases of diphtheria were thus exposed to the vapor. I have never been able to kill more than a small proportion of the bacteria in this way when they were taken from fresh, vigorous cultures. If the bacteria had been dried for a long time, so that they were in a feeble and vulnerable condition, it was found, in a few cases, that all had been destroyed by the vapor.

Another set of experiments was done, similar to those above described, save that the fabrics on which the streptococcus was previously dried were moistened just before the sulphur was burned in the receiver. This moistening, in one set of cases, was done by the direct application to the fabrics of sterilized distilled water; in another, by conveying condensed steam into the receiver, just before the sulphur was burned, until the water began to collect and run down the walls. Under these conditions, all the streptococci were killed within twenty-four hours.

A similar set of experiments on the action of sulphur vapor was made with the dried pseudo-membrane from cases of diphtheria. In these, I found that, whether dry or previously moistened, I could, in no case, succeed in killing all the streptococci by a twenty-four hours' exposure to the vapor. The difference of the action of the vapor on the pure cultures and on the streptococci enclosed in the pseudo-membrane is obviously due to the circumstance that, in the pure culture, there is direct contact between the acid and the germs, while in the membrane there is not.

We thus see that, while the moist sulphur vapor is more efficient than the dry in destroying the streptococcus diphtheriæ, in either form it is entirely inefficient for the purposes of practical disinfection.

Carbolic acid. In determining the germicidal power of this agent for the streptococcus diphtheriæ, a different mode of experimentation was adopted. A measured volume of actively growing beef-tea culture was intimately mixed, in sterilized tubes, with an equal volume of a freshly prepared aqueous solution of the acid. This, of course, would give the effect of a solution of one-half the strength of the carbolic solution employed, because it was diluted one-half by the fluid culture. In this way varying strengths of the acid were tested. As the length of exposure to the germicide is a most important factor in such tests, small portions of the mixture were removed from the tube, and instantly planted *in* gelatine, at the following intervals: ½, 3, 5, 10, 15 minutes. Approxi-

mately equal amounts were removed each time, by the use of a fine platinum wire loop. The portions removed were planted in gelatine tubes, by the Esmarch "roll" method, and in this way the number of colonies which appeared after from 3 to 5 days could readily be counted, and represented, of course, the number of germs which had survived the action of the germicide. In each case, a control-plant was made from the original culture, so that the number of germs destroyed could be approximately determined. Of course, absolute accuracy in determining the number of germs destroyed was neither sought for nor desired, but only such data as would indicate, in a general way, the efficiency of the germicide. I do not deem it necessary to reproduce here the numerical tables which embody the results of these experiments; I shall simply summarize the results. Cultures from several of the diphtheria cases were used, and the experiments repeated for control.

In $\frac{1}{2}$ per cent. solution of carbolic acid, the bacteria were slowly killed; but at the end of 15 minutes, about $\frac{1}{6}$ of the original number remained alive. In 1 per cent. solution, a large proportion were killed within 1 minute, and, after 3 minutes' exposure, none remained alive. In 2 per cent. solution, a total destruction occurred within 1 minute.

That it would be most unsafe to base rules for practical disinfection, however, upon the power of carbolic acid to kill the streptococcus diphtheriæ in pure cultures is shown by the following experiments on fresh pseudo-membranes. Small fragments, about 4 mm. square and 1 mm. thick, were put in 1 per cent. carbolic solution. Some of these were removed at the end of fifteen minutes, and planted; large numbers of streptococci grew, together with other forms. At the end of thirty minutes, plants showed that still a considerable number had remained alive. At the end of forty-five minutes all were dead. A similar series of experiments, with $2\frac{1}{2}$ per cent. carbolic acid, showed that within fifteen minutes all of the streptococci were killed. In much more dilute solutions of carbolic acid (1 : 4000), soaking of the pseudo-membrane for an hour and a quarter had no appreciable effect upon the vitality of the streptococcus.

We thus see that carbolic acid, in order to be of practical efficiency in destroying the streptococcus diphtheriæ, must be used in at least 2 per cent. solutions, and that its action must be prolonged.

Creolin. The experiments on the effect of this agent were made because of its highly lauded germicidal power, and because it has been claimed by many to be less harmful in its effects upon the organism than is carbolic acid, which in many respects it resembles. The preparation used was Merck's, and it forms with water in the strengths employed a milky, tarry-smelling fluid. The mode of experimentation was the same as with carbolic acid.

In solutions of 1 : 1000, about $\frac{1}{20}$ of the streptococci remained alive at

the end of 15 minutes. In solutions of 1 : 500, a considerable number remained alive at the end of 15 minutes. In 1 : 200 solutions, all were destroyed at the end of 15 minutes. In 1 per cent. solutions, all were dead at the end of 3 minutes. Thus, creolin in solutions of equal strength, appears to be a little more efficient in killing the streptococcus diphtheriæ than is carbolic acid.

Bichloride of mercury. The experiments with this agent were similar in character to those with carbolic acid and creolin.

In solutions of 1 : 5000, from $\frac{1}{6}$ to $\frac{1}{40}$ of the germs introduced as pure cultures remained alive up to the end of 1 minute. At the end of 3 minutes all were dead. In solutions of 1 : 2000, as well as 1 : 1000, complete destruction had occurred at the end of $\frac{1}{2}$ of a minute.

With the streptococci embedded in, and partially protected by, the pseudo-membranes, however, the effects are markedly different. Bits of pseudo-membrane, like those used in the carbolic acid experiments above, were soaked in solutions of varying strengths, with the following results: In 1 : 10,000, for 1 hour and 10 minutes streptococci grew in large numbers. In 1 : 5000, after 15 minutes a few grew; other fragments removed after 30, 45, and 80 minutes showed no growth. In 1 : 1000, after 15 minutes' soaking of the membrane a few streptococci grew. After this period, at intervals of 30, 45, and 80 minutes there was no growth.

We thus see that a fragment of diphtheritic pseudo-membrane must be soaked for from 15 to 30 minutes, in solutions of sublimate as strong as 1 : 1000, to accomplish a destruction of the streptococcus which in pure cultures, where there is perfect contact, may be achieved in half a minute.

I regard the numerical results of these experiments with germicides as of very little importance, save as giving general indications as to the absolute and relative power of killing the streptococcus, because, as already stated, of the great variability in the vigor of the germ from different cases and under a variety of conditions.

In the disinfection of objects, both the strength of the germicide and the length of exposure should be carried with large allowance beyond the point which such experiments may indicate as efficient. In the use of germicides in treatment, on the other hand, the limit will be usually sharply defined by the tolerance of the body to their poisonous effects.

Summary.

We have found, by a critical examination of the studies which have heretofore been made on diphtheria by the use of the modern methods of research, that no definite species of bacteria has been discovered which could fairly be positively regarded as the cause of the disease.

We have studied in detail, both by morphological and biological methods, twenty-four cases of diphtheria in young children, most of them occurring in hospitals, in which the affection has assumed the character of an epidemic, the disease either occurring by itself or in connection with suppurative diseases, or scarlatina, or measles. In all but two of these twenty-four cases, we have demonstrated the presence of a streptococcus, usually in large numbers in the local lesions, and in small numbers, in a few of the cases, in the viscera.

We have established, by the biological methods, the specific characters of this streptococcus, and by animal inoculations in rabbits and pigeons have found that it is markedly pathogenic, inducing erysipelatous or phlegmonous inflammation, abscess, and localized necrosis. We have not succeeded in inducing on the mucous membrane of animals any lesions which could fairly be regarded as similar to the local lesions of diphtheria, as these are ordinarily developed in man. We have found, however, that diphtheria as we see it in man, does not apparently occur spontaneously in animals, and has not been induced experimentally by any of the numerous and variously modified inoculations thus far practised.

We have in control examinations of mouth and tonsil scrapings from thirty-one healthy and sick children, not apparently exposed to diphtheria, never found the streptococcus, except in two cases of scarlatina in which diphtheria soon after developed. On the other hand, in examining throat and tonsil scrapings from forty children exposed to the disease in a hospital in which it was epidemic, we have found the streptococcus in twelve of them. In two of these, fatal diphtheria soon followed. Once we have found it floating in the dust of the room in which the disease had originated. We have seen that all of these observations, taken together, seem to lead us to so strong a presumption that the streptococcus is the causative factor, in this group of cases, at least, of diphtheria, that it practically amounts to a demonstration.

We have found reason for believing on biological and experimental grounds that the streptococcus occurring so constantly in these cases of diphtheria is probably identical with the streptococcus pyogenes and streptococcus erysipelatos. It has been shown that the apparent identity of the inciting species of bacteria in erysipelas, in some forms of phlegmonous inflammation and in at least certain groups of cases of diphtheria is not only not inconsistent with our knowledge of the pathology and symptoms of these three forms of inflammation, but serves to account for various facts concerning the nature and spread of diphtheria which have hitherto seemed obscure.

We have found that the crypts of the tonsils form a favorite nesting-place for the streptococcus and that it may lie harmlessly there even in considerable numbers, unless some lesion of the mucous membrane pro-

vides conditions suitable for its growth, when it may enter upon a career of active and, as it would seem, fateful proliferation.

In the presence of the streptococcus in large numbers in the local lesions of diphtheria and in its absence in any considerable number as a rule in the internal organs, we find ground for the belief that the symptoms of systemic infection are probably in large measure due to the absorption of a soluble poison produced by the bacteria at the seat of their most active proliferation.

We have demonstrated that the streptococcus of diphtheria is not readily destroyed by drying, but both in the form of pure cultures on threads and fabrics and in the pseudo-membrane itself may retain its vitality for long periods when dried in the air. We have tested its vulnerability when brought in contact with some of the commonly used antiseptic agents and found first that the vapor of burning sulphur is very inefficient and unreliable in destroying the germs. We have found that carbolic acid and creolin in considerable strength destroy the streptococci but in dilute solutions are inefficient; while sublimate even in very dilute solutions heads the list in its killing power. But we have also found, as was to be expected, that all of these germicides are much less efficient when applied to the bacteria which lie embedded in the pseudo-membranes and the tissues, than when they are free in the pure cultures.

Prophylaxis.

Having now accumulated a certain amount of definite knowledge about the germ which appears to be capable of inducing diphtheria, let us see how this knowledge may be applied in the prevention and in the treatment of the disease.

It is a well-established fact, to which the personal experiences of many a practising physician can bear witness, that diphtheria may be directly communicated from one individual to another by means of portions of the fresh pseudo-membrane. With this mode of transmission we have nothing to do. That it may be communicated in some less tangible way, either during the illness or long after, in garments, bedding, furniture, the walls of rooms, etc., is equally well established. But in the latter mode of infection our lack of knowledge about the nature of the disease germ has made it impossible for us to understand in detail its exact mode of transmission. In the light of these studies, however, this would seem to be tolerably plain.

We have seen that, in a certain group of cases at least, diphtheria may be caused by a streptococcus; that this streptococcus may retain its vitality for long periods when dried in fragments of the pseudo-membranes or in mucus. Now this dried material when detached and *broken* up forms a part of the dust of the room in which it is set free, and

like other dust may be inhaled. Exposed again to moisture, warmth, and such food as the mouth and respiratory passages abundantly furnish, the germs, as our experiments have shown, may revive and grow. Wherever then a particle of diphtheritic exudate dries on walls or garments or elsewhere we have apparently a possible source of infection—the conditions of its dissemination being those which equally control the spread of non-infectious dust. That drying destroys a certain proportion of the germs which a diphtheritic pseudo-membrane contains is shown by our experiments. How long a part of them may remain alive we do not know; but it appears from the experiments that the vitality as well as the virulence of the streptococci varies a good deal in different cases. That the germs cannot detach themselves from moist surfaces, such as the mucous membranes, and carry contagion through the expired air has been established by experiments on other infectious diseases.

We thus see that, save for those liable to come in contact with fresh diphtheritic exudates, the danger of infection seems largely to limit itself to exposure to dust and dirt of which the dried exudate forms a part. There seems to be every reason for believing that in the hospital from which most of our cases came, the dust infection was the common mode.[1]

This condition of affairs but emphasizes the probability that in the crusade against fifth and filth diseases, the devotees of cleanliness and prophylactic medicine will find in the immediate future a wide and widening field of labor in impressing upon the public the dangers of infectious dust in overcrowded houses and hospitals and filthy city streets and showing them the ways in which its evils may be avoided.

Already it has been shown that one of the most active sources of contagion in tuberculosis is the dust of rooms in which without due cleanliness consumptive individuals have lived. Antiseptic surgery has adopted such practical procedures as imply the belief that all dust-containing air may be infectious so far as wound diseases are concerned. There is much reason for believing that the pneumococcus is disseminated in the same way.

House disinfection. Having thus brought together the facts bearing upon the mode of communication of diphtheria which these studies embody, the practical deductions as to general sanitary precautions against the spread of the disease are comparatively simple. The immediate destruction so far as possible of all the inflammatory exudates which may be discharged is of primary importance. These should be received at once so far as possible into vessels containing 5 per cent. solution of carbolic acid, where they should remain for at least an hour before being thrown into the sewer. The removal of dust settled about

[1] As to the relationship of erysipelas and phlegmonous inflammation to diphtheria, as affording a source of infection, we need not dwell upon it here since the same conditions would seem to apply as in the transmission of the contagion from one case of diphtheria directly to another.

the room which may contain particles of the exudate not destroyed—by means of moist cloths, and never by that barbaric survival, the feather duster—and the efficient disinfection of clothing and rooms liable to contamination; these seem to be the other main points.

It is a great pity that in the matter of disinfection of rooms we should in this region still be going through with the inefficient mummery of burning sulphur with closed doors under the impression that it will destroy contagion. This operation has, indeed, a certain archaic picturesqueness about it, and save for the damage which is liable to accrue from the fading of furniture and hangings, is a tolerably harmless practice; but it savors rather of the propitiatory sacrifices to malevolent deities of centuries gone by than of the intelligence of the present time. Sulphurous acid, as it is usually applied in house disinfection, has been shown over and over again by the most careful experiments to be a very inefficient and unreliable disinfecting agent. It may be better than nothing, but in disinfectants—which are often our sole weapons in fighting epidemics—the best is none too good. The efficiency of sulphurous acid may be increased by securing the thorough wetting of everything to be disinfected, but even then it is not great.

As to the details of a mode of room disinfection upon which we may rely, I think it may not be out of place here to formulate a set of directions which largely embody, and are in part a transcript of the official regulations for disinfection as required by the health authorities in Berlin in 1887.

All bed-linen, clothing, handkerchiefs, etc., which are to be washed, and all cloths which have been used for dusting the room, should be placed for at least twenty-four hours in 2 per cent. solution of carbolic acid, then boiled for an hour in water, and then washed with strong soapsuds.

The disinfection of the room and its contents at the close of the illness will be the more easy, and certainly efficient, the greater the care which has been exercised in removing all unnecessary articles of furniture, hangings, pictures, etc., from it at the commencement of the disease.

In the first place, all clothing which cannot be washed, bedding, mattresses, pillows, etc., carpets, cushions, and all such furniture as has not exposed wooden frames, should be tied up in cloths (sheets) which are saturated with 2 per cent. carbolic solution, and sent away to be steamed at the public disinfecting station.[1]

All valueless articles of furniture or clothing should be burned—best at the disinfecting station—but in case of necessity the smaller articles

[1] A large and well-arranged disinfecting plant is now being planned for the city of New York, and will, it is hoped, soon be in operation. Where such disinfecting stations do not exist, as they should in every large town, a thorough scrubbing of the surfaces of mattresses, pillows, etc., with 2 per cent. carbolic solution and subsequent prolonged exposure to the air, and boiling of blankets, coverlets, etc., after washing in the carbolic solution, will be perhaps the best procedure.

may be disposed of in the house furnace or range at such time as cooking is not going on.

Polished articles of furniture, picture-frames, metallic articles, etc., should be firmly rubbed off on all their surfaces with dry cloths, or, when permissible, with cloths wet with 5 per cent. carbolic acid. The cloths used should be immediately burned or put into 2 per cent. carbolic solution.

If the walls of the room are hard-finished or painted, they, together with all doors, windows, and woodwork, should be thoroughly washed, as should finally the floor, with 5 per cent. carbolic solution. If the room have papered or frescoed walls, the floors should be thoroughly flushed with 5 per cent. carbolic solution, and then all the walls should be thoroughly and firmly rubbed down in every part with lumps of bread, the crumbs being allowed to fall on the carbolized floor. Then the woodwork is washed with carbolic solution, the crumbs gathered up and burned, and the floors washed with water.

The room should be finally exposed as fully as possible to the air for at least twenty-four hours, and longer if it is practicable.

Now, this will seem at first, no doubt, a very formidable process, but where a large public disinfecting plant is available it is, after all, comparatively simple; and, what is much more important, if faithfully and intelligently executed, will insure purification of the infected room. Of course, other germicides than carbolic acid might be used for washing the room, such as 1 : 1000 sublimate solution, but the experimental data which these studies furnish indicate that the carbolic solution will kill the streptococcus if it comes in contact with it.

Treatment.

That which these studies furnish in the way of suggestions for the treatment of diphtheria has already been indicated—at least for him who reads between the lines—in every fact set down regarding the life-history and vulnerabilities of the streptococcus and its relations to the varying phases of the disease. The obvious lesson taught by a definite conception of the nature of the germ which causes diphtheria is not to dally with fancy mixtures which have at best a moderate germicidal power, but to get at the growing germ, as directly as the seat of the lesion will permit, with some agent which we know will kill it.

It does not lie within the scope of this paper to suggest any detail as to the treatment of diphtheria. But the points which I would especially emphasize are: First, the evidence here adduced that the germ primarily gains a foothold at the seat of the local lesions, inducing its general effects upon the body at large by the local production of an absorbed ptomaine; second, the vulnerability of the germ to certain commonly employed germicides; third, the propriety of making germicidal applications to

the mouths and throats of individuals liable to infection. It will thus be seen that the immediate practical outcome of these studies, so far as the treatment is concerned, is rather to confirm the importance of local antiseptic methods, and to give precision to the ends which we may hope to attain by them, than the suggestion of anything new.

The writer hopes, in conclusion, that these studies, by lending clearness to our conception of its nature, may aid in the control as well as in the treatment of a disease which, in town and country, in this and other lands, claims yearly so many victims.

February 7, 1889.

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THE CONSTRUCTION

OF

O'DWYER TUBES,

WITH A

REPORT OF THREE HUNDRED AND FIFTY CASES OF INTUBATION OF THE LARYNX.

Read before the American Pediatric Society, held in New York City, June 3 and 4, 1890.

BY

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NEW YORK.

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THE CONSTRUCTION

OF

O'DWYER TUBES,

WITH

A REPORT OF THREE HUNDRED AND FIFTY CASES OF INTUBATION OF THE LARYNX.

SINCE the introduction of intubation in the treatment of stenosis of the larynx, by Dr. Joseph O'Dwyer, it has been my lot to see a great many cases, and it has been my good fortune to be intimately associated with its inventor during this time.

The object of this paper is to present some practical points derived from my experience, and to call special attention to the many badly-made tubes, and to the dangerous modifications of them, which not only cause permanent injury or death to the patient, but bring great disrepute upon one of the most brilliant and valuable operations of modern times.

The difficulty of the operation is often greatly underrated, and the importance of a wide experience in the treatment of these cases is seldom appreciated. Although it requires but a few seconds to insert the O'Dwyer tube, and the immediate results are most brilliant and gratifying, I know of no operation in surgery which is more brutal and shocking than the efforts of an unskilful man to intubate. With each miserable attempt, the child grows more frightened and more cyanotic, the face and clothes become smeared with blood, and, unless the operator is an unusually cool one, he loses his wits and

does fearful damage to the larynx. This picture is neither overdrawn nor rare, and I venture to state that the great majority of deaths which have been reported as due to pushing down membrane was the result of unskilled efforts, and due either to apnœa from prolonged attempts at introduction, or to asphyxia from forcing the tube through a false passage.

O'Dwyer and I have operated upon over six hundred cases, and neither of us have ever had a death on the table due to pushing down membrane. It may be true that some men find but little difficulty to intubate, even without previous practice on the cadaver, but this is certainly exceptional. It seems odd that a writer should say that "any surgeon possessing a knowledge of the anatomy of the parts and a good degree of surgical skill will be able to easily and quickly introduce the tube, and to remove it when the proper time arrives," in direct contradiction to a statement in the same article that in attempting to remove the tube, he "made a complete failure" and deferred the operation till next day. Another man writes that in the first case he was at work nearly half an hour before he succeeded in introducing the tube, but in the second case he introduced it in about five minutes. Ten seconds would be a liberal allowance of time in which to do this operation. Many other examples could be given both from the literature of the subject and from my own personal knowledge.

To understand the value of a large experience you must encounter some accident or unusual feature. When membrane is pushed down before the tube, or it is loose in the trachea or bronchi, how shall we get it out before the child chokes to death? Its importance is seen in this and many other questions which constantly arise, and the solution of which depends entirely upon individual judgment and experience.

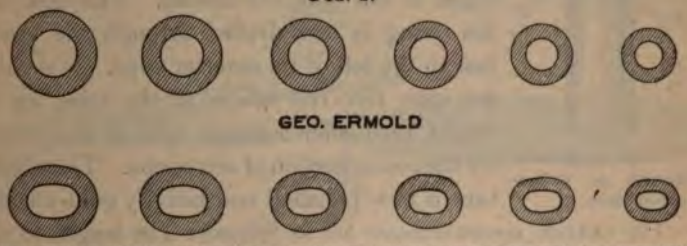
INSTRUMENTS.

There are three distinct sets of O'Dwyer tubes: one for children, containing six tubes; one for adults, containing ten tubes (although three tubes only are necessary for any form of *acute* stenosis in adults); and a third one, to be used only in children, for foreign bodies or loose membrane in the larynx. The set for adults differs from the well-known children's set

in size only; but the tubes for foreign bodies differ very materially from the others.

O'Dwyer tubes.—The small tubes are made of brass, and then heavily plated with gold. The adult tubes are made either of brass (gold-plated) or of hard rubber, each material having special advantages over the other. The details of their manufacture are as follows: Through a solid rod of brass, a hole of the proper size is bored, and then hammered on a mandrel, so that the outline is elliptical and the antero-posterior

FIG. 1.



Cross-section of tubes, natural size, before and after being hammered on a mandrel.

diameter of the opening is about twice its lateral diameter. It is thus made to conform to the shape of the larynx, and the greatest possible air-space is obtained with the least pressure upon the vocal cords. The next step is to file down the tube, so that it has the shape of a double wedge, the belly standing out laterally. By this means the tube is held in position and prevented from being coughed out. In taking away the extra metal, the walls of the tube should be made as thin as possible, except at the anterior surface of both the upper and lower ends, where considerable metal should be allowed to remain. This enables the instrument-maker to round off the upper end, which presses against the base of the epiglottis, and which would otherwise be sharp and cause ulceration at this point. It also allows him to round off the lower end and do away with the sharp, cutting edge, which would otherwise cause ulceration of the anterior wall of the trachea, from the up-and-down motion of the tube with each act of deglutition.

FIG. 2.

Lower end of tube, showing the rounded and thickened anterior edge.

No matter how carefully the upper end is made smooth,

there will be deep ulceration at the base of the epiglottis, due to pressure, if the tube is perfectly straight. This pressure is obviated by removing a wedge from the posterior edge of the tube, near the head, and, after knocking it back, it is firmly soldered in this position. The head, which is made in a separate piece, is now fastened over this. This head should be oval-shaped, and every part of it rounded off and smooth, so that there are no sharp edges to cut into the tissues. The hole for the string is now drilled through the head, just to the left of its anterior edge. It should not open into the calibre of the tube, for it would then offer a rough spot as a nucleus for the accumulation of secretions. The whole tube is now polished and heavily gold-plated.

FIG. 3.

Tube in rough, after removal of wedge-shaped piece.

The outside measurements are as follows: The length of all the adult tubes are the same,—namely, three inches. The lengths of the croup-tubes are $2\frac{5}{8}$, $2\frac{1}{2}$, $2\frac{1}{4}$, 2, $1\frac{3}{4}$, and $1\frac{1}{2}$ inches, respectively; the antero-posterior diameters are $\frac{12}{32}$, $\frac{11}{32}$, $\frac{10}{32}$, $\frac{9}{32}$, $\frac{8}{32}$, and $\frac{7}{32}$ of an inch; and the width at the point of greatest swell, $\frac{12}{32}$, $\frac{11}{32}$, $\frac{10}{32}$, $\frac{9}{32}$, $\frac{8}{32}$, and $\frac{7}{32}$ of an inch, respectively.

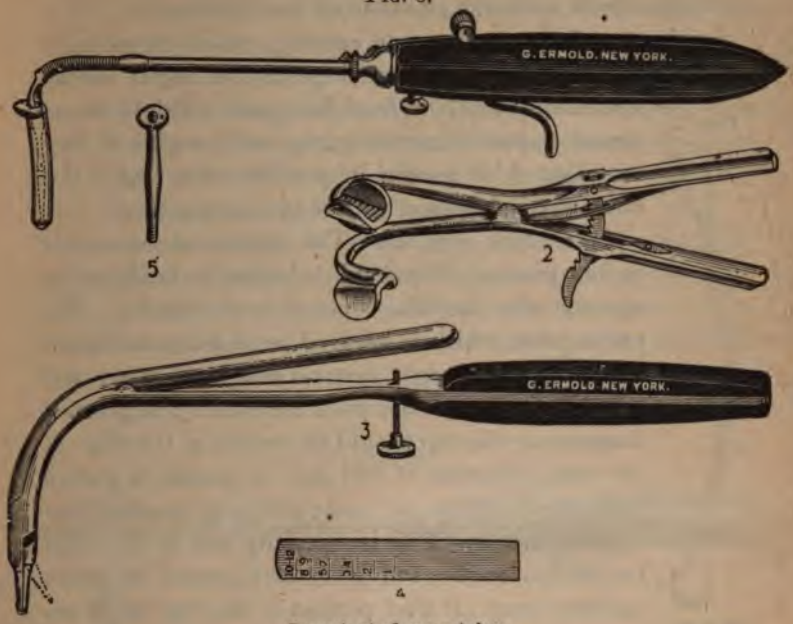
FIG. 4.

Head of tube.

The tubes for foreign bodies and loose membrane in the trachea and bronchi are made of very thin German silver, and are all short and of the same length,—namely, $1\frac{1}{8}$ inches. The head is small; there is no retaining swell, because the tube is held in position by pressure from its large size, and the upper end of the tube is not thrown away from the epiglottis by taking out a wedge-shaped piece, because it is designed to let it remain in the larynx a few hours only. For the same reason, there is no objection to its lower edge being sharp, although during insertion the obturator should make a perfect probe-point with the tube. This set consists of seven tubes. They are cylindrical, but slightly smaller in calibre at their lower end. The diameter of the smallest one is $\frac{7}{32}$ of an inch, and there is an increase of $\frac{1}{32}$ of an inch with each tube. These

tubes are intended to fill the larynx to its full sub-glottic capacity; and it is confidently believed that any foreign body which

FIG. 5.



Correct set of croup-tubes.

could enter the larynx would be able to pass out through one of them, except those that increase in size by the absorption of

FIG. 6.

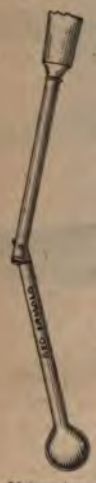
Set of tubes for foreign bodies or loose membrane in trachea and bronchi.

moisture. In the management of loose membrane in the trachea and bronchi, it is the most satisfactory method that I am famil-

iar with; and, if some means can be devised for detaching and breaking up the partly-adherent membrane, I believe that it will give us the solution of this problem,—the most difficult that forces itself upon the attention of the intubator.

In the use of these tubes, one warning should be strongly emphasized, namely, that they are kept in the larynx not by a retaining swell, but from their great width by direct pressure upon its narrow parts, and therefore if they are allowed to remain in position more than a few hours deep ulceration will be the consequence.

FIG. 7.



Obturator.

Obturators, gag, etc.—The obturators are made in four pieces. The shank is jointed to facilitate its removal after the tube is placed in the larynx. The probe point, which is soldered to its lower end, must fit the tube so snugly that no rough edges are left to catch membrane and push it down. The greatest diameter of this tip should fit exactly at the edge of the tube, otherwise it will fail to present a perfect probe-point, and it will cause difficulty in withdrawing the inserter. This is especially true of the tubes for foreign bodies, and great care should be taken to scoop away all that portion of the tip which remains within the body of the tube. The head of the obturator should fit the tube for some little distance to prevent it from wabbling antero-posteriorly.

The O'Dwyer *gag* has been modified by Denhard, Dennison, Sajous, and many others, but the simplest and most satisfactory is that of Denhard, the construction and use of which can be seen in the figure. The *scale* is an arbitrary measure, which enables the operator to select the proper tube. The tube which reaches the line marked 1 is intended for a child up to that age; the tube which reaches the line marked 2 is intended for a child up to that age; and the same for the lines marked 3 to 4, 5 to 7, and 8 to 10. The largest tube is intended for children who are more than ten years old but have not reached puberty. Of course, much individual judgment is sometimes called for in the selection of the proper tube to use, and it may be necessary to take a larger or smaller tube than the one indicated by the scale.

The *thread* should be strong and made of braided silk. The twisted silk is apt to unravel so that, when it is withdrawn, it gets caught and pulls the tube out with it.

The *introductor* is usually made correctly, its construction being very simple. It can be taken apart and cleaned very easily. The only difference between the one for children and for adults is the size. However, in the set for foreign bodies it has a longer curve, and, instead of the two flanges on either side which push off the tube, it has a single one behind, which runs along a groove in the head of the obturator.

The construction of the *extractor* is rather complicated, but, with a proper one for a model, the maker should experience no great difficulty in copying it. Three very important points should be remembered. *First*, the jaw which moves when the instrument is opened should be the one nearest the operator, while the opposite jaw is a continuation of the handle. This

FIG. 8.

greatly facilitates the removal of the tube, owing to its shape and direction. *Second*, when the jaws are closed, the end of the instrument should not be sharp, but should present a blunt probe-point, otherwise it will stick into the soft laryngeal tissues, and, besides making a false passage, will do great damage when it is opened. *Third*, no instrument should ever be used which does not have a regulating screw in the handle. It prevents the jaws from opening wider than is necessary, and it is of the greatest importance, even when the instrument is used by the most expert operator. If the jaws are allowed to

open to their full extent in the larynx of a small child, much damage will be done even if the instrument is not withdrawn. Of course in the latter case the injury is much greater, and it is possible in this way to split open the cricoid, so as to allow the tube to sink down deeply into the larynx, and might result in an abscess or some more serious complication.

Membrane extractors.—Many ingenious devices have been used for the removal of loose membrane from the trachea and bronchi; but the problem remains unsolved. With the exception of the new foreign body tubes of O'Dwyer, every attempt in this direction has been an absolute failure, and even these tubes are only a step in the right direction. The apparently insurmountable obstacles which prevented the practical realization of Dr. O'Dwyer's ideas have been overcome by him, one after the other,—to prevent ulceration of the cords, to prevent the expulsion of the tube by coughing, to prevent ulceration at the base of the epiglottis and at the lower end of the tube, and the many purely mechanical problems which were constantly presenting themselves. More recently, the difficulty in feeding after intubation was solved by Dr. Casselberry, of Chicago, by the very simple means of posture, after many unsuccessful attempts with a complicated and often dangerous artificial epiglottis, or with various changes in the shape of the head of the tube. The power to get rid of loose membrane in the trachea and larger bronchi will probably be obtained by means of the foreign body tubes, the link which is wanting being some method of breaking up the membrane and freeing it from its attachments in the bronchi.

FIG. 9.

Tube with jointed head, which enables a trained nurse to remove it quickly in case of emergency.

The illustration shows a tube devised by Dr. O'Dwyer, which enables the trained nurse to quickly remove the tube with the finger in case of emergency. Its practical value has not been fully tested, and fortunately the necessity for its use is not frequently met with.

Faulty tubes.—It is an impossibility, as far as O'Dwyer's experience and mine go, for an instrument-maker to copy a perfect tube unless the details of its construction are explained to him; and after this explanation, it is only with the greatest care, and after many failures, that they succeed. Therefore it is not surprising that the great majority of the tubes which are in use are imperfect and crude. Most of them will certainly do harm, and many of them will involve the life of the patient in great danger. These seem like strong terms, but they must seem very mild to those who understand the vast importance of having all the small details in the construction of the tubes made perfect, and who know how the country is being flooded with a great number of crude and improperly-made instruments.

The most common defect is the failure to throw the head of the tube away from the epiglottis by removing a wedge-shaped piece from its posterior edge. This neglect will positively cause ulceration, even to complete perforation, at the base of the epiglottis. Other common faults are rough points on the inner walls which form nuclei for the accumulation of secretions; a sharp lower edge which cuts into the anterior wall of the trachea with each act of deglutition; sharp edges on the head; and the failure to round off the upper and anterior portion of the tube.

The tube which I now show you was obtained from a physician, who returned to Mr. Ermold a perfect set of instruments, and sent this beautiful specimen as a model of the kind of tubes which he was accustomed to use and which he desired. It seems to possess every possible fault in the construction of an O'Dwyer tube and still retain a resemblance to one. It is a crude specimen of work, the lower end is sharp, the body of the tube is perfectly straight throughout its length, the anterior portion of the head is not rounded off, the hole for the thread opens into the calibre of the tube, and the edges of the head itself are nearly as sharp as a knife. This type of instrument is unfortunately not rare, and it is no exaggeration to say that the majority of tubes that are used should never be placed in a larynx.

It is a curious fact that, as far as I know, every modified

O'Dwyer tube has radical defects, and that every real improvement in the tube itself has emanated from the original inventor. These sketches give at a glance the evolution from the first tube (a simple wire frame, which was used only on the cadaver) to the shape now in use. It will be seen that the

FIG. 10.

Evolution of the tube.

principle of almost every so-called new modification had been previously tried and found wanting.

The Stoerck tube is perfectly straight, it is rounded off neither below nor above, and is held in position not by a retaining swell, but by its great pressure against the sides of the larynx. The probe-point of the obturator is drilled with holes, and the advantage claimed for it is that the child can

breath during the time of introduction. When we remember that it takes only a few seconds to do the operation, this rather theoretical gain is more than counterbalanced by its very glaring faults.

The Sajous modification has never been used in a living subject, otherwise it would never have been presented to the public. The bivalve principle was extensively tested by O'Dwyer, and it was found impossible to prevent such a tube from clogging up rapidly, usually in a couple of hours, and never longer than twenty-four. This is a serious defect, which would always count against any number of advantages, especially when they are purely theoretical. The modification of Dr. Thilo possesses the same fault, and, in addition, it is cylindrical.

Dr. Hoadley's suggestion, to insert the tube so that the posterior flange comes anteriorly, is certainly wrong; and deep ulceration at the base of the epiglottis will be the constant result. Even with a straight tube this will take place, as has been frequently demonstrated in the dead-house. The "deep-tubing" of the same writer, as done with modified tubes, has much to recommend it, if it overcomes the difficulty of swallowing. However, this is not so important, since Dr. Casselberry proved that this difficulty is absolutely obviated by posture. Hence, its advantage hardly compensates for the increased amount of pressure on the anterior portion of the larynx, and for the increased danger of the laryngeal tissues swelling over the top of the tube and obstructing it. The cup-shaped head, to facilitate the extraction of the tube, seems to be an excellent idea. O'Dwyer used a funnel-shaped head very early in his experiments, but abandoned it, as he thought that it offered greater facility for the entrance of fluids in drinking. The question of long or short tubes can be settled only by experience. Each has its advantages in individual cases; but I am convinced that the long tube is preferable, for the reason that any loose membrane in the trachea is less liable to obstruct it (from a valve action), and, also, the long tube is retained more easily.

Tasher's tubes have no advantages that I can see, and have three serious drawbacks. The head and the lower end of the tube is sharp and presents a cutting edge, which will do dam-

Cases of Intubation.

Date.	No.	In the practice of	Sex.	Age.	Duration of laryngeal symptoms.	Duration of diphtheria.	Urine.	Cause of death.	Complications.	Result.
Nov., 1889.	301.	John H. Ripley.	F.	8 yrs.	3 days.	6 days.	Albumen.	Extension to bronchi.	.....	Died 2½ days after intubation.
Dec., 1889.	302.	W. J. Burnett.	F.	2 yrs. 8 mos.	3 days.	Pres.	Not examined.	Exhaustion.	.....	Died 2 days after intubation.
Dec., 1889.	303.	J. E. Claussen.	M.	5 yrs.	2 days.	Pres.	Albumen.	Extension to bronchi.	Measles.	Died 2½ days after intubation.
Dec., 1889.	304.	J. Lewis Smith and S. H. Dessau	M.	15 mos.	2 days.	Pres.	Albumen.	Extension to bronchi.	Rickets.	Died 1 day 7 hours after intubation.
Dec., 1889.	305.	C. E. Young and W. A. Fanning.	F.	16 mos.	2 days.	None.	Not examined.	Extension to bronchi.	.....	Died 1½ days after intubation.
Dec., 1889.	306.	G. D. Spor.	F.	4 yrs. 3 mos.	6 days.	7 days.	Albumen.	.....	.....	Recovered; wore tube 5½ days.
Dec., 1889.	307.	G. V. Hann.	M.	4 yrs. 1 mo.	4 days.	6 days.	Albumen.	Sudden heart-failure.	.....	Died 3 days after intubation.
Dec., 1889.	308.	W. Freudenthal and F. de Kraft.	M.	4 yrs.	4 days.	Pres.	Not examined.	Extension to bronchi.	Extension to bronchi.	Died 7 hours after intubation.
Dec., 1889.	309.	W. J. Burnett.	M.	16 mos.	3 days.	Pres.	Not examined.	.....	.....	Recovered; wore tube 7 days.
Dec., 1889.	310.	W. A. Fanning.	M.	17 mos.	5 days.	7 days.	Not examined.	Asphyxia without tube.	.....	Died 1 day after intubation.
Jan., 1890.	311.	G. A. Hayunga.	F.	6 yrs. 3 mos.	4 days.	10 days.	Albumen.	Extension to bronchi.	.....	Died 2½ days after intubation.
Jan., 1890.	312.	Neil O. Fitch.	F.	3 yrs. 5 mos.	2 days.	5 days.	Not examined.	Extension to bronchi.	.....	Died 4 days after intubation.
Jan., 1890.	313.	W. J. Burnett.	M.	13 mos.	1 day.	None.	Not examined.	Sudden heart-failure.	Measles.	Died 4 days 5 hours after intubation.
Jan., 1890.	314.	A. G. Gerster and C. F. Kremer.	M.	4 yrs.	2 days.	Pres.	Albumen.	Nephritis.	.....	Died 3 days after intubation.
Jan., 1890.	315.	J. Lewengood and A. T. Swan.	M.	5 yrs. 6 mos.	5 days.	7 days.	Albumen.	Extension to bronchi.	.....	Died 3 days 8 hours after intubation.
Feb., 1890.	316.	Leo Schnepp.	M.	13 mos.	2 days.	Pres.	Albumen.	Extension to bronchi.	.....	Died 3 days after intubation.
Feb., 1890.	317.	C. F. Kremer.	F.	2 yrs. 6 mos.	2 days.	7 days.	Albumen.	Nephritis; convulsions.	.....	Died 2 days after intubation.
Feb., 1890.	318.	G. A. Hayunga.	M.	5 yrs.	1 day.	5 days.	Albumen.	Slow heart-failure.	Sepsis.	Died 1 day after intubation.
Feb., 1890.	319.	J. Lewis Smith and F. M. Warner.	F.	17 mos.	2 days.	3 days.	No albumen.	Pneumonia.	....	Died 5 days after intubation.
Feb., 1890.	320.	C. E. Young and P. E. Tiemann.	F.	2 yrs. 7 mos.	3 days.	Several days.	Albumen.	.....	.....	Recovered; wore tube 16 days 2 hours.
Feb., 1890.	321.	S. B. Allen.	F.	7 yrs.	2 days.	4 days.	Albumen.	Slow heart-failure.	Sepsis.	Died 1 day after intubation.
Feb., 1890.	322.	J. Lewis Smith and Joseph Anderson.	M.	3 yrs. 6 mos.	5 days.	Pres.	Albumen.	.....	.....	Recovered; wore tube 5 day 22 hours.

Mar., 1890.	323.	S. S. Jones.	F.	9 yrs. 6 mos.	1⅗ days.	Pres.	Albumen.	Extension to bronchi.	.....	Died 3⅕ days after intubation.
Mar., 1890.	324.	J. Harvie Dew.	F.	7 yrs.	1 day.	3 days.	Albumen.	Sepsis and extension.	Extension and sepsis.	Died 12 hours after intubation.
Mar., 1890.	325.	A. Jacobi and Leo Schnepp.	F.	3 yrs.	4 days.	.....	Albumen.	Pneumonia.	.....	Died 2 days after intubation.
Mar., 1890.	326.	H. Kolb.	F.	4 yrs.	5 days.	None.	Albumen.	Extension to bronchi.	....	Died 4 days after intubation.
Mar., 1890.	327.	H. H. Curtis and J. T. Kennedy.	M.	7 yrs.	2 days.	3 days.	Not examined.	.....	.....	Recovered; wore tube ½ hour.
Mar., 1890.	328.	J. W. Stronach.	F.	4 yrs.	4 days.	8 days.	Albumen.	Extension and sepsis.	.....	Died 2½ days after intubation.
Mar., 1890.	329.	H. G. Lyttle.	M.	3 yrs. 3 mos.	2 days.	Pres.	Albumen.	Sepsis.	.....	Died 4 days after intubation.
Mar., 1890.	330.	J. W. Stronach.	F.	22 mos.	1 day.	Pres.	Not examined.	Cerebro-spinal meningitis.	Cerebro-spinal meningitis.	Died 2 days after intubation.
Mar., 1890.	331.	Edward Sanders.	F.	5 yrs.	1⅗ days.	Pres.	Not examined.	Exhaustion.	Measles.	Died 1 day after intubation.
Mar., 1890.	332.	E. J. Ware and H. Z. Pratt.	M.	2 yrs.	1 day.	7 days.	Albumen.	Slow heart-failure.	.....	Died 18 days after intubation.
Mar., 1890.	333.	S. S. Jones and C. W. Van Wert.	M.	9 yrs.	1 day.	None.	Not examined.	.....	.....	Recovered; wore tube 1 day.
Mar., 1890.	334.	E. M. Moffett.	F.	5 yrs. 6 mos.	4 days.	Pres.	Albumen.	Extension to bronchi.	.....	Died 1½ days after intubation.
April, 1890.	335.	Charles Schram.	M.	14 mos.	3 days.	None.	Albumen.	.....	Pneumonia.	Recovered; wore tube 19 days.
April, 1890.	336.	R. H. Buck.	F.	3 yrs. 7 mos.	4 days.	None.	Albumen.	Sudden heart-failure.	.....	Died 1 day after intubation.
April, 1890.	337.	C. E. Young.	F.	3 yrs. 9 mos.	5 days.	7 days.	Albumen.	Sepsis.	Sepsis.	Died 3⅕ days after intubation.
April, 1890.	338.	G. V. Hann.	F.	2 yrs.	2 days.	None.	Albumen.	Extension to bronchi.	Scarlet fever.	Died 4 days after intubation.
April, 1890.	339.	E. W. Derby.	F.	7 yrs.	1½ days.	None.	Not examined.	.....	....	Recovered; wore tube 5½ days.
April, 1890.	340.	J. C. Schmincke.	F.	28 yrs.	3 days.	5 days.	Albumen.	Extension to bronchi.	.....	Died 2 days after intubation.
April, 1890.	341.	M. M. Dunton.	M.	8 yrs. 6 mos.	4 days.	14 days.	Albumen.	Extension to bronchi.	Extension.	Died 12 hours after intubation.
May, 1890.	342.	J. A. McLoughlin.	M.	4 yrs.	2⅕ days.	Pres.	No albumen.	Asphyxia.	.....	Died 2 days after intubation.
May, 1890.	343.	R. B. Burton.	F.	6 yrs.	3 days.	None.	Albumen.	.....	Pneumonia.	Recovered; wore tube 6 days.
May, 1890.	344.	W. J. Gardineer.	F.	4 yrs.	5 days.	Pres.	Albumen.	.....	.....	Recovered; wore tube 6 days.
May, 1890.	345.	Henry Schroeder.	M.	2 yrs. 7 mos.	1 day.	3 days.	Albumen.	Extension and sepsis.	Sepsis.	Died 1 day after intubation.
June, 1890.	346.	My own.	F.	2 yrs. 4 mos.	4 days.	Pres.	Albumen.	.....	Scarlet fever.	Recovered; wore tube 6 days.
June, 1890.	347.	G. V. Hann.	M.	5 yrs.	1 day.	1 day.	Albumen.	Extension to bronchi.	.....	Died 2 days after intubation.
June, 1890.	348.	J. A. Roth.	F.	22 mos.	11 days.	Pres.	Albumen.	.....	.....	Recovered; wore tube 16 days.
June, 1890.	349.	M. M. Dunton.	F.	6 yrs.	2 days.	6 days.	No albumen.	.....	.....	Recovered; wore tube 5½ days.
Oct., 1890.	350.	J. R. Conway.	F.	2 yrs.	3 days.	7 days.	Not examined.	Extension to bronchi.	Extension.	Died 1 day after intubation.

age; the laryngeal tissues will swell over its top on account of its deep position; and, the most important defect of all, it is held in place not by a retaining-swell, but by pressure against the narrow parts of the larynx.

The artificial epiglottis, which was first devised by Waxham, and has since been copied by others, increases the danger of asphyxia, both by presenting a nucleus for the accumulation of secretion, and by acting as a valve, which cuts off inspiration. Its usefulness was destroyed as soon as it was shown that a child could swallow perfectly well after intubation, if the head is placed in such a position that the pharynx is lower than the larynx. I believe that Waxham has abandoned its use.

It is frequently asked why the tubes cannot be cast, thereby reducing the price and obtaining uniformity in size and shape. It is impossible to do this in one piece, on account of the backward direction which the calibre takes near its upper end. Ermold has succeeded in casting a tube in two pieces, which are soldered together. However, it was found necessary to abandon their use on account of the rough spots or cracks which appeared in the tube, and formed nuclei for the accumulation of secretions. Even with the greatest care these defects could not be avoided.*

This makes a total of three hundred and fifty cases, with one hundred recoveries, or 28.5 per cent. The details of cases 1 to 200 were published in the *New York Medical Journal* for March 9, 1889; and of cases 201 to 300, in the *American Journal of the Medical Sciences* for 1891. During the period of time covered by the present report—namely, from November, 1889, to June, 1890—I saw six cases which recovered without operation. They occurred in the practice of Dr. W. J. Burnett, Long Island; Dr. C. E. Young, Dr. R. M. Cramer, Dr. W. A. Hawes and Dr. S. Baruch, Dr. D. F. King, and Dr. Joseph O'Dwyer. Two cases died without operation, one with Dr. J. A. McLoughlin and one with Dr. W. A. Hawes. Three cases died before my arrival, one with Dr. W. A. Fan-

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* Ermold has apparently solved this problem in the most ingenious manner, but it is too early to make any positive statements.

ning; one with Dr. DeWitt Hitchcock and Dr. B. G. Strong; and one with Dr. J. E. Kelly and Dr. J. J. Henna. It should be remembered that this does not represent the real number of patients which recovered without operation, but only the percentage of those cases in which the stenosis became severe enough to cause alarm and warrant a consultation to determine the advisability of operative interference.

"The Kensington,"
102 East Fifty-seventh Street.

THE SURGICAL TREATMENT

OF

ERYSIPELAS:

A SUCCESSFUL CASE IN AN INFANT.

Read before the Pediatric Section of the New York Academy of Medicine, May 8, 1890.

BY

DILLON BROWN, M.D.,

NEW YORK.

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were apparently due to headache. The rectal temperature was 104.5°, and the pulse, which was very weak and thready, was 178. The child was seemingly going into a condition of collapse, and we felt that it was a hopeless case unless the extension of the disease could be stopped.

Under chloroform anæsthesia, the Kraske-Riedel fence was made with a scalpel in the healthy tissues above the erysipelas and completely encircling the thigh. These scarifications were dressed with gauze wet with a solution of bichloride of mercury (1 to 1000). Both before and after the operation the thigh was thoroughly scrubbed with a solution of the same strength. Two hours later the site of the operation was marked by a red, elevated, angry band, which seemed to be erysipelatous, but which, probably, was due to the irritation of the bichloride of mercury. The inflammation rapidly travelled into the scarified area, but did not pass above this barrier. The next morning the baby was better in every way. Temperature 100°, pulse 120 and stronger, eyes brighter, and she took considerable notice of her surroundings. In three days there was no anxiety in regard to her condition, and she made a rapid recovery.

Studies on the Etiology of the Pneumonia Complicating Diphtheria in Children.

BY

T. MITCHELL PRUDDEN, M. D.,

Director of the Laboratory of the Alumni Association of the College of Physicians and Surgeons, N. Y.,

AND

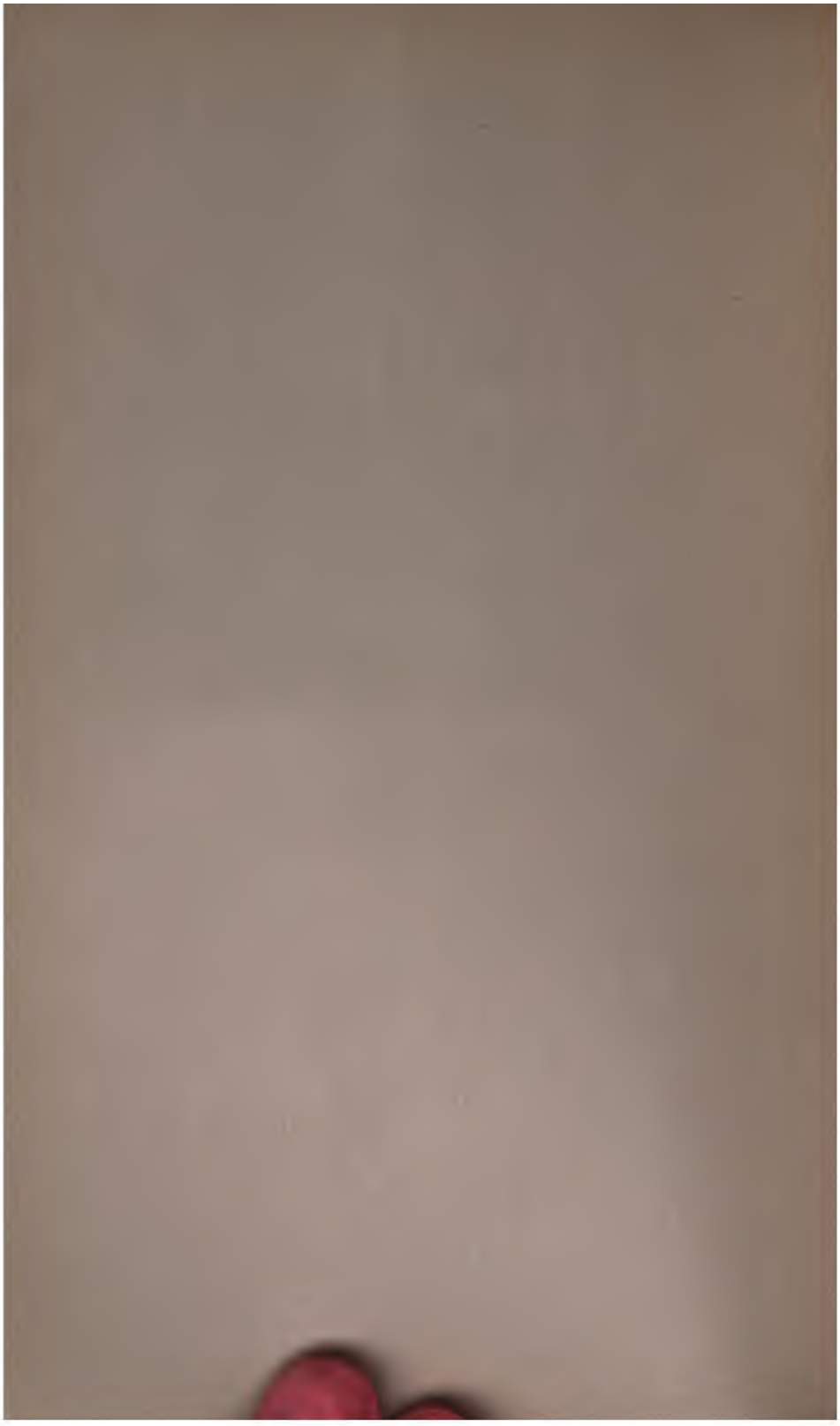
WILLIAM P. NORTHRUP, M. D.,

Pathologist to the New York Foundling Asylum.

FROM

THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES,

JUNE, 1889.



Extracted from the
American Journal of the Medical Sciences for June, 1889.

STUDIES ON THE ETIOLOGY OF THE PNEUMONIA COMPLICATING DIPHTHERIA IN CHILDREN.[1]

By T. Mitchell Prudden, M.D.,
DIRECTOR OF THE LABORATORY OF THE ALUMNI ASSOCIATION OF THE COLLEGE OF PHYSICIANS AND SURGEONS, N. Y.,

AND

William P. Northrup, M.D.,
PATHOLOGIST TO THE NEW YORK FOUNDLING ASYLUM.

The classification of the inflammations of the lungs has assumed a new phase since the improved methods of studying the bacteria have become definitely formulated and widely practised. While formerly morphological distinctions, either gross or minute, between the different forms of inflammation were ardently sought after and minutely detailed, the pathologist is now disposed, while still cherishing the approved morphological data, to seek for the etiological factors which more or less definitely determine the varying phases which inflammations of the lungs may assume, and upon these to base, at least, a provisional and supplementary working classification.

Thus it has been found that that common and well-defined form of pulmonary inflammation called acute lobar pneumonia is, in a large proportion of cases at least, associated with and no doubt directly induced by the growth in the lungs, when the conditions are favorable, of a diplococcus known as the Diplococcus pneumoniæ of Fränkel and Weichselbaum. This bacterium, as appears from the researches of Sternberg, Fränkel, and others, is a not uncommon denizen of the mouths of healthy persons.

In the new etiological classification of inflammations of the lungs, then, acute lobar pneumonia is an inflammation usually induced by the Diplococcus pneumoniæ, and presenting in general those clinical gross and minute features which have long been known and frequently described. This form of pneumonia, while so frequently lobar in its extent and distribution as to justify its name, may, nevertheless, develop in various

1 The statistical data on the frequency of the disease, the clinical history of the cases, and most of the children's lungs made use of in these studies have been furnished by Dr. Northrup. The microscopical and experimental work was done by Dr. Prudden.

atypical ways, and occurring, as it sometimes does, in lobular form has still been found to be associated with the same bacteria. Whether in inciting this disease the bacteria always find access to the lungs through the upper air-passages, as seems most probable, or occasionally from the blood, we cannot yet positively say.

While, furthermore, it seems well established that the Diplococcus pneumoniæ is the most common primary bacterial etiological factor in the ordinary acute lobar pneumonia, it is not unlikely that some other forms of bacteria, particularly the pneumo-bacillus of Friedländer, may occasionally incite the disease and cause typical symptoms and lesions.

Another form of inflammation of the lungs, protean in its phases, is that which occurs directly or indirectly under the influence of the Bacillus tuberculosis. That the bacteria of tuberculosis, in a great many cases at least, gain access to the lungs with dust in the inspired air, is well established.

Still another form of pulmonary inflammation, as viewed from the etiological stand-point, is the so-called embolic pneumonia induced by the lodgement in the pulmonary bloodvessels and their subsequent growth in the lung tissue of bacteria, more commonly the pyogenic cocci, brought from some other part of the body.

In these well-defined forms of pulmonary inflammation the inciting species of bacteria have been identified by cultures over and over again by various observers, so that their nature as bacterial diseases may be regarded as well established. All of these inflammations are liable to be associated with bacteritic inflammations due to the same cause in various other parts of the body (1).

In sharp distinction to these bacteritic pneumonias stands a group of lesions, generally considered more or less loosely as inflammations, which, although sometimes remotely and secondarily, are never directly dependent upon the presence of bacteria in the lungs. Such are the chronic interstitial and intra-alveolar pneumonias, be they anthracotic or cardiac or syphilitic in origin, or due to trauma or some other local cause, such as foreign bodies, tumors, etc. We do not now speak of gummata of the lungs, of the relations of which to bacteria we are still in ignorance.

It is probable that the ordinary so-called hypostatic pneumonias are not regularly associated with the presence of bacteria. The part which bacteria play either primarily or secondarily in gangrene of the lungs is too little known to permit of a definite classification of its varying phases here. It seems hardly necessary to mention here that the lesions of the bacteritic and the non-bacteritic inflammations of the lungs are very frequently associated in the same individual.

While definite and positive researches by the improved culture methods have justified us in such a grouping as indicated above of certain of the more common bacteritic, in distinction from the non-bacteritic pneu-

monias, there is still a considerable number of phases of the inflammatory process in the lungs, especially those large and important groups known as the secondary lobar or lobular and broncho-pneumonias about the direct inciting cause of which we as yet know very little.

These phases of inflammation are apt to occur under a great variety of conditions as complications of other disorders. While we know a good deal about the conditions under which they most frequently occur, the bacterial studies upon them by the modern methods have been so few and so desultory that it is at present impossible to say definitely whether bacteria do or do not stand in a causative relation to many or most of them.

In acute lobar pneumonia, the clinical history, lesions, and complications of which are so well defined and typical, the presumption is decidedly against more than one species of bacteria being the common and ordinary inciting cause. On the other hand, in the lobular and broncho-pneumonias, both of adults and children, if they are bacterial in origin at all, the presumption appears rather in favor of the hypothesis that there may be many species of bacteria which may serve as the inciting cause of the lesion under different circumstances.

If we make a brief summary of the definite results which have been obtained by the bacterial studies on the secondary complicating lobular and broncho-pneumonias, rejecting for the most part the numerous simple morphological examinations, which are, from the nature of the case, always incomplete, and considering only those which by the culture methods have led to decisive results, we shall find that while a good beginning has been made, the observations are, on the whole, very incomplete.

That localized inflammations of the lungs may be induced by the presence of anthrax bacilli, by the bacillus of glanders, and by the actinomycosis is well established, though in man in this region these forms of pulmonary inflammation are not frequent enough to be of great practical importance.

There is a form of pneumonia which is a direct propagation of the inflammatory process from the pleura (pleurogenous pneumonia) and which has been proven to be due to the same organisms as those inciting the primary pleurisy.

There are, finally, some isolated observations, not sufficiently numerous and not fully enough controlled by the culture methods to be regarded as very significant, which would indicate the probability that now and then the Bacillus typhosis and the Streptococcus erysipelatos may, in the course of typhoid fever and erysipelas, obtain a foothold in the lung and induce a more or less extensive lobar or lobular inflammation (2).

In 1886 Weichselbaum (3), in his studies on various forms of acute pneumonia, found a streptococcus in eight cases of secondary pneumonia.

Two of these were lobar and associated one with rheumatism and the other with senile gangrene of the leg. The others were lobular, and associated with erysipelas, phlegmonous inflammation of the nose and mouth, progressive paralysis, and nephritis. This streptococcus appeared to him on both morphological and biological grounds to be similar to the Streptococcus pyogenes and Streptococcus erysipelatos, though he does not positively declare his belief in their identity. He names it the Streptococcus pneumoniæ.

Neumann (4) has cultivated from the lungs in a case of simple broncho-pneumonia the Staphylococcus pyogenes aureus and albus.

In 1887 Guarnier (5) found in the lungs of a child dead of broncho-pneumonia after measles, by morphological and culture methods, a streptococcus apparently identical with the Streptococcus erysipelatos.

In connection with his studies on the etiology of diphtheria in 1884, Löffler (6) made morphological examinations of the lungs of several persons dead of diphtheria. In the typical hemorrhagic broncho-pneumonia of this disease he frequently found streptococci, and not the bacilli which he had found in the pseudo-membrane. As he made no cultures, however, from the lungs, these observations are not of great significance.

In 1885 Fränkel (7) cultivated from the lung in a case of broncho-pneumonia complicating diphtheria in an adult, a streptococcus which seemed to him morphologically and biologically identical with Streptococcus pyogenes.

Experiments on the artificial induction of lobular and broncho-pneumonia in animals by the introduction of bacteria into the lungs have been made by several observers, and will be considered later in this paper.

It would thus appear from the scattered results which we have gleaned in the literature that the logical procedure in studying the etiology of the secondary lobular and broncho-pneumonias is not to make the attempt at first to investigate every form of the disease which comes to hand under the greatest variety of conditions, but rather to attempt the systematic study of certain well-defined groups or classes of the lesion. One of the best defined, as well as one of the most important of these groups, is the broncho-pneumonia which so frequently complicates measles and scarlatina and diphtheria in children.

In connection with recent studies by one of us on the etiology of diphtheria in children (8) we have made a series of observations and experiments on the broncho-pneumonia which so frequently complicates the disease which it is the purpose of this paper to record.

Frequency of the Occurrence of Broncho-pneumonia in Diphtheria.

Darier (9) in 181 autopsies upon patients dead of diphtheria found broncho-pneumonia in 61 cases.

Talamon (10) in 121 autopsies found broncho-pneumonia in 69 cases.

Schrakamp (11) in 54 autopsies found broncho-pneumonia in 31 cases.

In 195 autopsies upon children dead of diphtheria at the New York Foundling Asylum, well-marked, recent broncho-pneumonia was present in 133 cases.

If we reduce these data to percentages, we see that Darier found broncho-pneumonia in 34 per cent. of cases of fatal diphtheria, Talamon in 64 per cent., Schrakamp in 57 per cent., and Northrup in 68 per cent. On the whole, then, we may say that so far as these statistics go, broncho-pneumonia is present in fatal diphtheria, as a complication, in about 55 per cent. of the cases.

These data, however, have but a limited significance, since a careful microscopical examination of the lungs of children dead with diphtheria of the air-passages shows that there may actually be a considerable amount of lobular inflammation without gross change. Thus, in the 195 New York Foundling Asylum cases there was a considerable number of lungs in which there were areas of localized congestion or hemorrhage or collapse, which a microscopical examination might have shown to be associated with slight inflammatory changes, but which, owing to the uncertainty, were not counted among the pneumonias. This condition of affairs would seem to indicate that the above percentage is low rather than high.

Seventeen Cases of Diphtheritic Broncho-pneumonia Examined.

These cases have been described more fully in the paper on diphtheria above referred to, so that here only those details are given which bear directly on the subject in hand. But it should be borne in mind that, as already recorded, cultures as well as microscopical examinations were made of the pseudo-membranes in all of them, and the Streptococcus diphtheriæ was found, usually in large numbers, in all except Case XIV.

Cases I. to XIV., inclusive, were from the New York Foundling Asylum. Cases XV. and XVI. were from the Randall's Island Infant Asylum. Case XVII. from private practice.

Case I.—Male, three years two months. Diphtheria following rectal abscess; death on fifth day. Autopsy: Voluminous firm pseudo-membrane in larynx, trachea, and larger bronchi. Moderate amount of broncho-pneumonia, with large involvement of bronchi. *Bacterial examination of lungs:* Small numbers of cocci and diplococci in bronchial

exudate as well as in the air-spaces. *Cultures* showed large number of *streptococci;* a few Staphylococcus pyogenes aureus and a few stout, round-end bacilli with scattering forms.

CASE II.—Male, seven months. Diphtheria following pertussis; death on fourth day. Autopsy: Moderate amount of pseudo-membrane in trachea and larger bronchi; this in the bronchi was much softened. Broncho-pneumonia, involving right lower and part of middle lobe. *Bacterial examination of lungs:* Large numbers of cocci in bronchial exudation, and enormous numbers, single and in chains, in exudation in air-vesicles. *Cultures* show vast numbers of *streptococci* and a few Staphylococcus pyogenes aureus.

CASE III.—Female, two and one-half years. Diphtheria; death on seventh day. Autopsy: Pseudo-membrane in pharynx, larynx, and trachea; old and fresh broncho-pneumonia in both lower lobes posteriorly. *Bacterial examination of lungs:* In areas of old broncho-pneumonia no bacteria were found. In the fresh bronchial exudation, as well as in areas of fresh and commencing consolidation, enormous numbers of cocci, single, paired, and in chains. *Cultures* from areas of fresh broncho-pneumonia showed great numbers of *streptococcus* colonies and a few of Staphylococcus pyogenes aureus.

CASE IV.—Male, three years. Diphtheria following pertussis, with albuminuria and broncho-pneumonia on third and fourth day; death on eighteenth day after commencement of pertussis. Autopsy: Softened membrane in large and small bronchi, with considerable old and moderate amount of fresh broncho-pneumonia. *Bacterial examination of lungs:* No bacteria found in areas of old consolidation. In the bronchial exudate, as well as in the areas of fresh consolidation, large numbers of cocci, single, double, and in chains, with a few scattering forms. *Cultures* show large numbers of *streptococci* from bronchial exudate and from areas of fresh consolidation; also a few Staphylococcus pyogenes aureus.

CASE V.—Young child. Diphtheria, followed by broncho-pneumonia, accompanied by umbilical phlegmon. Autopsy: Voluminous membrane in larynx, trachea, and bronchi; congestion and broncho-pneumonia in both lower lobes. *Bacterial examination of lungs:* Enormous numbers of cocci, single, paired, and in chains, in bronchial exudation and in contents of air-vesicles in consolidated areas. *Cultures* show vast number of *streptococci* and considerable number of Staphylococcus pyogenes aureus.

CASE VI.—Young child. Diphtheria, followed by broncho pneumonia. Autopsy: Moderate amount of pseudo-membrane, very slight amount of fresh broncho-pneumonia, considerable amount of old broncho-pneumonia. *Bacterial examination of lungs:* In bronchial exudation few cocci and scattering bacilli; very few cocci in areas of commencing consolidation in lungs. *Cultures* show moderate number of colonies of *streptococci*, considerable number of Staphylococcus pyogenes aureus, and many stout bacilli.

CASE VII.—Young child. Diphtheria with umbilical phlegmon. Autopsy: Pseudo-membrane in larynx, pharynx, and trachea, loosening and softening below. Considerable amount of fresh broncho-pneumonia in both lower lobes. *Bacterial examination of lungs:* In exudation in air-spaces, largely made up of red blood-cells, enormous numbers of streptococci. *Cultures* show large numbers of *streptococci*, Staphylococci pyogenes aureus, and a considerable number of scattering forms.

CASE VIII.—Female, three years eight months. Diphtheria following measles; death on sixth day. Autopsy: Softening membrane in trachea, extending into bronchi. Both posterior lower lobes red and mottled with solid areas. *Bacterial examination of lungs:* Considerable atelectasis with a little exudation in scattered regions. The smaller bronchi in the affected regions are filled with pus-cells, and desquamated epithelium, mingled with a large number of cocci, single, paired, and in chains. In the air-spaces adjacent and belonging to these affected bronchi the air-vesicles are filled with cellular and granular exudation, intermingled with cocci, single and in chains. *Cultures* show large numbers of *streptococci*, with a few Staphylococcus pyogenes aureus and some scattering forms.

CASE IX.—Male, two and one-half years. Diphtheria; death on eighth day. Autopsy: Pseudo-membrane in pharynx and larynx; no broncho-pneumonia evident to the naked eye. *Bacterial examination of the lungs:* Acute bronchitis with considerable exudation in some of the smaller bronchi. Large numbers of streptococci mingled with the exudation in the lumen of the inflamed bronchi. In the bronchioles, air-passages, and air-vesicles nearest to the inflamed bronchi and belonging to the same systems were enormous numbers of streptococci and single cocci. In the air-vesicles a few desquamated epithelial and pus-cells and granular material were intermingled with the bacteria. In this case we seem to have a very early stage of the broncho-pneumonic process. *Cultures* show considerable numbers of *streptococci* and a few scattering forms of bacilli.

CASE X.—Female, one year. Diphtheria of pharynx and tonsils; enterocolitis; death on third day. Autopsy: Small amount of firm pseudo-membrane on pharynx, tonsils, and at the upper end of œsophagus. Both posterior lower lobes of the lungs show congestion and a moderate amount of scattered lobular consolidation. *Bacterial examination of lungs:* Consolidated areas in the lungs are mostly due to old broncho-pneumonia with much exudation in the smaller bronchi. In fresh exudation a few cocci were found intermingled with the cells, while in the areas of old consolidation none were discovered. *Cultures* from the redder areas of pneumonia showed a few *streptococci*, a few Staphylococcus pyogenes aureus and albus, and some scattering forms.

CASE XI.—Female, two years. Broncho-pneumonia with apparent recovery, followed by diphtheria of the pharynx and fresh broncho-pneumonia, with death on second day. Autopsy: Irregular consolidation of the left lower lobe with congestion. *Bacterial examination of lungs:* The broncho-pneumonia was found to be largely old, but in places the exudation was fresh, accompanied by considerable congestion. In the areas of old consolidation no bacteria were found. In the air-spaces containing the fresh exudation a considerable number of streptococcus chains were found. *Cultures* from the lungs showed large numbers of *streptococci*, a considerable number of Staphylococci pyogenes aureus, and a few scattering forms.

CASE XII.—Male, three years. Diphtheria following scarlatina, tubercular peritonitis; small patch of pseudo-membrane at the base of the tongue and in the larynx and trachea; softening below. Autopsy: Both lungs showed scattered areas of congestion and partial consolidation. *Bacterial examination of lungs:* Microscopical examination of the lung showed that the consolidated areas were in large part due to the old pneumonia, while in the congested parts there was a moderate

amount of fresh exudation and enormous numbers of streptococci, often forming large masses of snarls or chains. *Cultures* from pneumonic areas in both lungs showed vast numbers of *streptococci*, considerable numbers of Staphylococcus pyogenes aureus and albus, with a few stout bacilli.

CASE XIII.—Diphtheria, with death on third day. Autopsy: Pseudo-membrane in the pharynx, larynx, trachea, and bronchi; firm and voluminous. Moderate amount of consolidation in both lower lobes of the lungs. *Bacterial examination of lungs:* Very few scattering cocci were found in consolidated areas and in bronchial exudation. *Cultures* of consolidated areas in the lung show a large number of *streptococci* (pure culture).

CASE XIV.—Male, three and one-half years. Enterocolitis, diphtheria, intubation; death on third day. Autopsy: Moderately firm pseudo-membrane in the larynx; small amount of broncho-pneumonia in both lower lobes. *Bacterial examination of lungs:* Moderate amount of exudation in spots in the lungs, but only a very few scattering forms of bacteria, mostly stout bacilli. *Cultures* from the lungs showed considerable numbers of *streptococci*, and only a very few scattering forms.

CASE XV.—Diphtheria following measles; death on seventh day. Autopsy: pseudo-membrane in larynx and trachea; tubercular bronchial glands; consolidation of both lower lobes of the lungs; pleurisy. *Bacterial examination of lungs:* Considerable old broncho-pneumonia. In new exudation a moderate number of cocci. In areas of old broncho-pneumonia no bacteria were found. *Cultures* of the lungs show a large number of *streptococcus* colonies, a few of Staphylococcus pyogenes aureus, and scattering forms.

CASE XVI.—Diphtheria following measles; death in twenty-four hours. Autopsy: No pseudo-membrane; commencing broncho-pneumonia. *Bacterial examination of lungs:* Moderate number of cocci, single and in chains, in the exudation. *Cultures* show many *streptococcus* colonies and a few scattering forms.

CASE XVII.—Female, two and one-half years. Diphtheria with early development of constitutional symptoms, tracheotomy, death. Autopsy: Voluminous pseudo-membrane in trachea; slight broncho-pneumonia. *Bacterial examination of lungs:* Microscopical examination not made, the lungs being destroyed by mistake after the cultures were started. *Cultures* of pneumonic areas of the lungs show large numbers of *streptococcus* colonies and besides these only a few scattering forms.

SUMMARY OF EXAMINATIONS AND CULTURES FROM DIPHTHERITIC BRONCHO-PNEUMONIA CASES.

We thus see that the areas of fresh broncho-pneumonia in the lungs of seventeen young children, dead of diphtheria, showed, with one exception, the presence of considerable or large numbers of streptococci. The exceptional case was one in which no streptococci were found in the pseudo-membranes. The streptococcus was the only form present in the pneumonic areas with such uniformity and in such abundance as to appear of significance. The streptococci isolated from the lungs in these cases present exactly the same morphological, biological, and pathogenic characters as those of the streptococci found in the pseudo-membranes

from the same cases, and which have already been described and called Streptococcus diphtheriæ (8).

The reason for believing these bacteria to be identical with the Streptococcus pyogenes and Streptococcus erysipelatos are set forth in the paper above referred to.

Control Cases.

Non-diphtheritic Broncho-pneumonia.—For purposes of control I have made cultures from the consolidated areas in the lobular and broncho-pneumonia of young children, not associated with diphtheria (10 cases). In two of the cases the pneumonia occurred with whooping-cough, in one with erysipelas, in two with marasmus, in five without any other apparent complication. In only a few of these ten cases have I found either by microscopical examination or by cultures any species of bacteria in considerable numbers, nor were the same species found in the different cases with sufficient frequency to indicate that, whether abundant or not, they were of special significance. Thus in two of the cases there were in the pneumonic areas many of the short bacilli which are common in the mouths of children, and which I have called chain-bacilli, and in four of the cases there were very small numbers of Streptococcus pyogenes aureus. The streptococcus was found in only one of these cases.

This exceptional case, the only one out of ten of non-diphtheritic broncho-pneumonia examined in which streptococci, both by culture and microscopical examination, were shown to be present in the lungs, was the above-mentioned case of erysipelas. In this case, however, although the cultures showed large numbers of streptococci in the lungs as well as in the skin, liver, kidney, and spleen, in the lungs the bacteria were only found in the bloodvessels, and never in the exudation in the bronchi or air-spaces. In this case, then, there was a general infection through the bloodvessels with erysipelas streptococci, and, although present in the bloodvessels of the lungs, they apparently have no relation either in situation or in numbers to the slight amount of broncho pneumonia present.

Acute Lobar Pneumonia.—Furthermore, I have made cultures from three cases of typical acute lobar pneumonia in children between two and three years old. These children all died at an early period in the disease, and from the consolidated areas in all of them I cultivated enormous numbers of the pneumococcus of Fränkel and Weichselbaum. In addition to this species, in one of the cases there were large numbers, in another a few, of the Staphylococcus pyogenes aureus. Besides these there were only scattering forms. No streptococci were found in these cases.

Normal Lungs.—Finally, I have made cultures from lungs, apparently normal, taken from the bodies of children dead of enterocolitis (two

cases), marasmus (three cases). In none of these lungs have I found either microscopically or by cultures, streptococci. In two of the cases of marasmus, the common fluidifying fluorescent bacillus of water was present in considerable numbers, and in the others only a few scattering forms.

Summary of Cultures in the Control Cases.

In ten cases of lobular and broncho-pneumonia in children not diphtheritic, in three cases of typical acute lobar pneumonia in young children, and in five cases dead of enterocolitis or marasmus with apparently healthy lungs (eighteen cases in all), the cultures of the lungs showed no streptococci, except in one case of erysipelas with general systemic infection; in six a few Staphylococcus pyogenes aureus were present. Besides, there were a few scattering forms, mostly short, stout bacilli, in the various cases.

General Summary of Cultures in all the Cases.

We thus see that streptococci were present in large numbers in the freshly affected lobular and broncho-pneumonic areas of children suffering from diphtheria in fifteen out of sixteen cases; while in the lungs either normal or pneumonic of eighteen non-diphtheritic children placed under the same sanitary conditions no streptococci were found except in the bloodvessels of a case of erysipelas with general infection.

Histological Details.

In recording the results of the morphological and biological examination of the lungs in these seventeen cases of pneumonia associated with diphtheria, and placing them in contrast with similar results obtained from the lungs in non-diphtheritic cases, it has seemed wise to give only a succinct statement of the facts elicited. Some histological data have, however, been developed in the various examinations which are not without significance in connection with the general subject of lobular and broncho-pneumonia in young children.

In the first place, in many of the control cases of simple lobular and broncho-pneumonia, of which that subacute or chronic form which is so common in marasmus children may be regarded as typical, neither by cultures nor by morphological examinations can we find evidence that bacteria are present or have anything to do with the lesions. In this form of pneumonia the bloodvessels in the affected regions are apt to be dilated, the walls of both bronchi and air-spaces more or less thickened, and often infiltrated with cells. While the air-spaces contain sometimes a few, sometimes many, large cells, which seem to be desquamated alveolar epithelium, neither blood nor fibrin nor pus is commonly present. This form of pneumonia sometimes is and sometimes is not

associated with atelectasis. It appears to correspond in its general nature to some forms of hypostatic pneumonia in adults. Now, in many of our cases of diphtheria occurring in the New York Foundling Asylum this form of lesion was the prominent, and in many of the early fatal cases, was the only lesion evident in the gross examination.

On the other hand, I have found in the lungs of nearly all of the diphtheritic children, sometimes with and sometimes without the above form of lesion, an entirely different type of inflammation. In this inflammation in its well-marked phases there sometimes is and sometimes is not congestion of the bloodvessels in the affected regions. The smaller bronchi are apt to be more or less blocked with pus cells, and the air-spaces to contain in varying amounts pus-cells, red blood-cells, fibrin, granular material, desquamated epithelium, and bacteria. The bacteria in our cases were almost exclusively cocci, and the larger proportion of these were arranged in streptococcus form. These bacteria did not appear to be generally enclosed in the exudation cells, though often clustered close about them; nor were they often found in the walls of the air spaces, but they lay free in the exudation mass or were clinging to the walls of the vesicles. They were present sometimes in very small numbers, but often in enormous quantities.

Not infrequently only a few small areas in the lungs were involved in this way, and these were more frequently in the posterior portions. When markedly hemorrhagic in character, so that the air-spaces contain large numbers of red blood-cells, the involved areas in the gross examination of the lungs appear like areas of localized intense congestion, but they are apt to be lobular in distribution. Atelectasis was rarely observed in the regions involved in this form of pneumonia.

While in the main there is such a degree of inflammation in the smaller bronchi as to render the designation of broncho-pneumonia applicable to the lesion, there are apt to be in most of the lungs areas of consolidation in which no change in the bronchi can be made out. Such areas of simple lobular inflammation may constitute the entire lesion in some lungs without any participation of the bronchi.

The distribution and general characters of this lesion, whether involving the bronchi or not, would seem to indicate that it is a form of inspiration pneumonia, "Schluck-pneumonie" of the Germans. This view is sustained by the observation that it was apt to be most marked and highly developed in children which had died after the pseudo-membranes in the air-passages above had begun to soften and break down.

Not infrequently one encounters in these lungs larger and smaller areas of consolidation in which the exudation is exactly of the same character as that just described, and indicative of an acute inflammatory process, but in which bacteria are entirely absent or, at least, not to be observed by any of the various means commonly resorted to. The explanation

of this condition will be seen when we come to the results of animal experiments.

A very marked feature of this form of secondary broncho- or lobular pneumonia with diphtheria is the hyperplasia of the lymph nodes and nodules in and about the lungs. Not only are the larger and smaller bronchial lymph nodes (lymph glands) usually greatly enlarged, but the minute islets of lymphatic tissue (lymph nodules) which are scattered about the lungs in the interstitial tissue, as described by Arnold, are, especially in and near areas of inflammation, so large as to form prominent features in the microscopical sections.

Some of the varying phases of this form of pneumonia and the relation of the tissue to the streptococci are shown in the plates.

In Plate I. is represented a small bronchus from Case IX., in which the epithelium is nearly all detached and, together with mucus, a few pus-cells, and streptococci, forms an exudation mass partially filling the lumen of the tube. The air-vesicles adjacent to this bronchus and belonging to its system of air-spaces contained large numbers of pus and epithelial cells intermingled with streptococci.

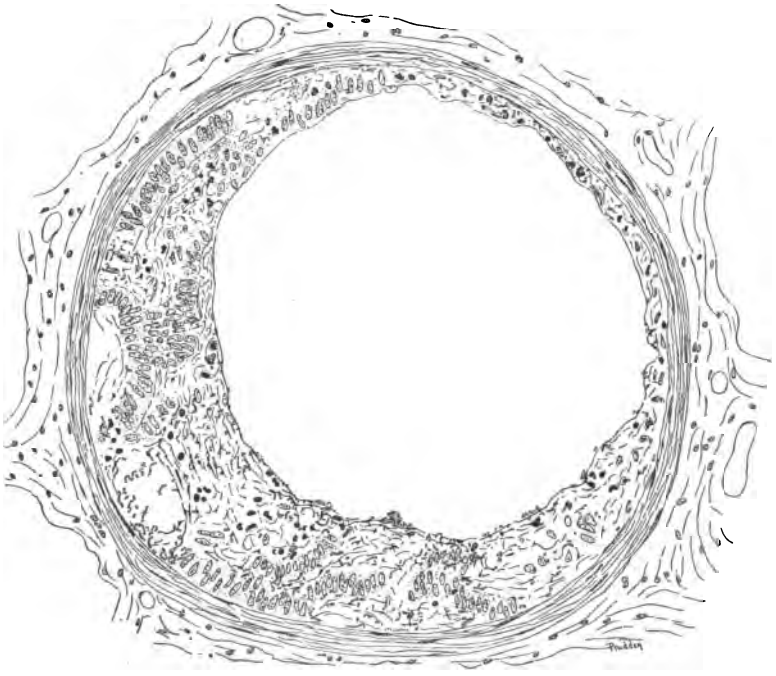
Plate II., Fig. 1, shows two adjacent air-vesicles in consolidated areas of the lungs of Case V. In one of these vesicles the inflammatory exudate is largely fibrinous, while in the other there are epithelial and pus-cells. In both the streptoccoci are present in considerable numbers.

Plate II., Fig. 2, shows three adjacent air-vesicles from the lungs in Case VII., in which the exudate is largely made up of red blood-cells and granular material. Here the streptococci are present in very large numbers.

It should be mentioned here as bearing upon the theme of this paper, that the air of the New York Foundling Asylum while these researches were in progress, was usually found to contain the Staphylococcus pyogenes aureus and albus, and in one analysis the streptococcus floated with the dust. Furthermore, the bacterial examination of the scrapings of the mouths and throats of children, both sick and well, who were inmates of the asylum showed that the Staphylococcus pyogenes aureus was frequently present in those situations in considerable numbers. While the Staphylococcus pyogenes was present in the lungs in a considerable number of the cases of broncho-pneumonia with diphtheria, the lack of uniformity in its occurrence and the small numbers in which it was usually found, coupled with its frequent occurrence in the air and in the mouths of healthy children, would lead us to infer that its presence in the lungs in these cases is of but doubtful significance.

Our observations thus far, then, would seem to justify us in the hypothesis that if the acute broncho-pneumonia which accompanied the diphtheria in the cases which we have examined was bacterial in origin at all, it was probably due to the entrance into the lungs through the

PLATE I.

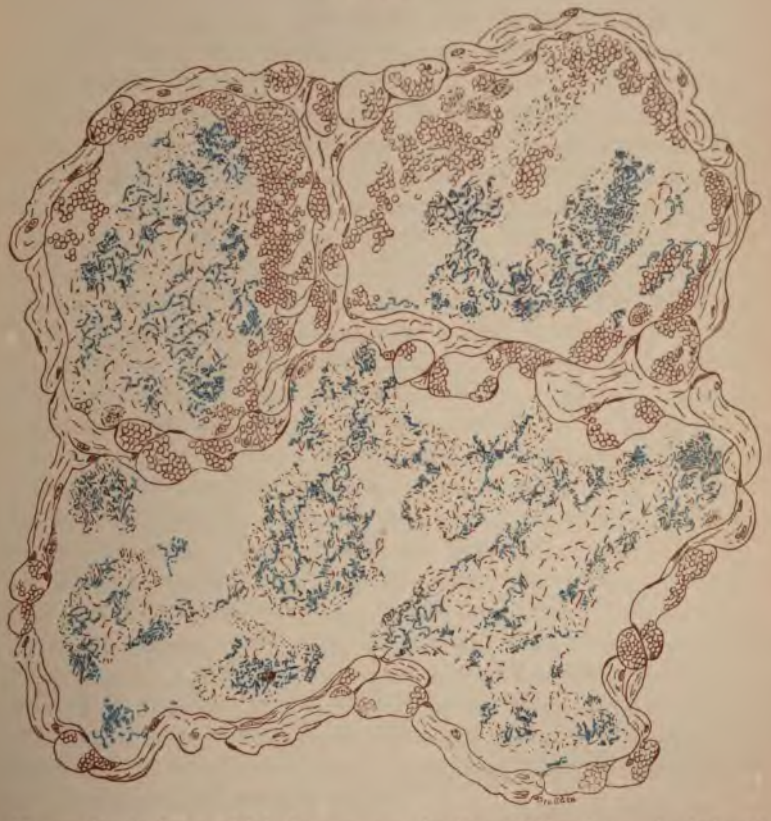


Small branches from CASE IX. Showing exudation with streptococci.

PLATE II.—FIG. 1.

Two adjacent air-vesicles from CASE V. Showing exudation consisting of fibrine, leucocytes, and epithelium, intermingled with streptococci. (The bacteria are drawn proportionally a little too large.)

FIG. 2.



Hemorrhagic form of lobular pneumonia, CASE VII. Intermingled with *the red blood cells and* granular material in the air vesicles are large numb *of streptococci.*

bronchi from the local lesion above of the Streptococcus diphtheriæ. But plausible as this hypothesis appears to be, it is necessary to have recourse to animal experiments in order to complete its demonstration. To these let us now turn our attention.

Animal Experiments.

A good deal of experimental work has been done in the artificial production of lobular and broncho-pneumonia in animals, either by the introduction of irritating vapors or fluids or solids directly into the lungs through the trachea, or by causing the entrance into the air-passages of fluids and solids from the mouth as the result of section of the vagus nerves. To the results of these experiments I need only thus briefly refer, since our theme is limited in scope to the introduction of pure cultures of known forms of bacteria.

I have used in these experiments, which were done exclusively on rabbits, cultures of the streptococci obtained from the lungs of five of the above described cases of diphtheritic broncho-pneumonia. Actively growing beef-tea cultures from two to five days old were used.

Preliminary experiments have shown that the introduction of the streptococcus directly into the lung by a puncture of the thoracic wall, is usually followed by fibrinous pleurisy and localized inflammation of the lung about the point of puncture. But the conditions of infection in this way are so much more complex and so unlike those under which the broncho- and lobular pneumonia of diphtheria in children occurs that the details of my experiments in this direction need not be given here.

Injections of the Streptococcus Diphtheriæ into the Lungs of Rabbits through the Trachea.—The mode of procedure in these experiments was to sterilize carefully the skin about the neck, after cutting away the hair, and then having made a small incision, exposing the trachea, to thrust the needle of the injecting syringe through its walls. From one to five c.c. of the beef-tea culture was injected, the small wound washed with sublimate or carbolic solution and bound up. The animals were held upright for a few moments after the injection in order to allow the fluid to run down into the lungs. Usually there was little or no local inflammatory reaction at the point of operation in the neck. Fifteen rabbits were operated upon in this way.

In most cases immediately after the injection coarse râles were heard over the thorax of the animals and in about one-third of the cases there was marked dyspnœa. Three of the animals with symptoms of increasing dyspnœa, loss of appetite, and progressive weakness, died, two on the second and one on the third day after the operation. The remaining animals appeared to recover rapidly from the initiatory dyspnœa, and afterward showed no sign of illness. These were killed at intervals of from three and one-half hours to ten days.

In seven of the fifteen cases no gross lesions were seen in the lungs. In the remaining cases there were larger or smaller areas of localized consolidation and congestion grouped in lobular form about the larger and smaller bronchi. In none of the cases was there evidence of any lesions of the other viscera.

If the animals were killed early—within the first twelve hours—although no gross changes, or, at most, localized areas of moderate congestion were seen, the streptococci could not only be cultivated from the lung tissue, but they were readily seen with the microscope, after staining by Gram's method, scattered here and there in considerable numbers in the air-vesicles, in the larger air-spaces, and in the bronchioles. They lay in part free in these situations, and in part were clustered about the alveolar epithelium. It was not easy to decide whether the epithelium actually contained the bacteria or whether they simply lay on the surface. I have not been able, even as early as three and a quarter hours after the injection of the streptococci into the trachea, to find any of them in bronchi large enough to be lined with ciliated epithelium.

A microscopical examination was made of the lungs of the rabbits which were either killed or died, at the following intervals after the intratracheal injection of the streptococcus, 3½, 12, 20, 24, 28, 40, 60 hours, and 3, 4, 8, 10 days. This examination showed in general that in four out of the seven lungs in which no gross lesions were visible, there was actually a considerable amount of lobular inflammation with more or less bronchitis, and that the same condition prevailed in the lungs which showed gross lesions. But in the latter cases the inflammatory lesions were more extensive and were usually associated with congestion, thickening of the walls of the air-vesicles, more or less atelectasis, and swelling of the smaller bronchial lymph nodes.

Cultures were made in the usual way. In no case were streptococci found in the viscera other than the lungs, and after from twelve to twenty-eight hours they were found in every case to have disappeared from the lungs also.

Before twelve hours I have been unable to find any evidence of inflammation save here and there a moderate congestion of the small veins and capillaries of the pulmonary air-spaces. In animals which died or were killed in from twelve to sixty hours after the injection I have failed to find streptococci either by microscopical examination or by cultures.

The degree of inflammatory lesion in these animals varies considerably in the different cases. The smaller bronchi may contain pus and the epithelium be loosened and desquamated. The most marked and constant change is the swelling and proliferation of the alveolar epithelium in circumscribed regions. Sometimes only a small cluster of air-vesicles is involved in this way; sometimes the change is to be observed over areas of considerable extent. The air-vesicles are sometimes filled nearly

full of new cells, which, from the karyokinetic figures which are to be observed in the nuclei (Plate IV. Fig. 1), I conclude to be derived from the alveolar epithelium. With these larger cells are mingled varying numbers of pus-cells and often a little fibrin (see Plate IV. Fig. 2). The adventitia of the smaller veins in certain regions is apt to be infiltrated with leucocytes (Plate IV. Fig. 1), and the lymph nodules about the smaller bronchi to be much swollen and prominent (Plate III.).

In animals killed after this period—sixty hours—when any lesion at all was found there was apt to be the same kind of exudation in the air-spaces, but the walls of the air-spaces and the smaller bronchi appear thicker than normal, and sometimes with, sometimes without, atelectasis; the lung was dense and firm in patches.

SUMMARY.—It would thus appear that the most common effect of the injection of a moderate amount of the Streptococcus diphtheriæ into the lungs of rabbits through the trachea is to cause a well-marked broncho- and lobular pneumonia with hyperplasia of the lymph nodules; that the bacteria disappear early from the lungs, and the pneumonia in many cases is slight in extent and evanescent; but that in a few cases the inflammation is prolonged by an involvement of the walls of the air-spaces.

INJECTION OF THE STREPTOCOCCUS DIPHTHERIÆ INTO ANIMALS' LUNGS WHICH WERE ALREADY THE SEAT OF INFLAMMATION.—In order to learn the effects of the streptococcus when introduced into lungs which were already in an inflamed condition, I have induced in another series of animals both mild and severe inflammation of the bronchial mucous membrane by the preliminary introduction of a few drops either of strong or very dilute solutions of ammonia through the trachea. This was followed at varying intervals by the introduction of two to three c.c. of a beef-tea culture of the streptococcus.

It is difficult in these experiments to decide how much of the broncho-pneumonic lesions which are almost invariably observed when the animals are killed after from twenty-four to twenty-seven hours are due to the ammonia and how much to the streptococci. For the preliminary experiments on ammonia injections alone showed that a broncho-pneumonia very similar in character to that caused by the streptococci alone was almost always induced. The streptococci, of course, formed no part of the exudation in the air-spaces in the cases in which ammonia alone was used. In the animals which had received both ammonia and streptococci, however, the lesions are more marked and extensive, and the bacteria did not disappear so early from the lungs as when the inflammation was induced by them alone.

This fact would seem to furnish another illustration of the well-established principle that certain pathogenic bacteria maintain their existence

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TREATMENT OF

SCARLET FEVER

AND

ITS COMPLICATIONS.

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TREATMENT OF

SCARLET FEVER AND ITS COMPLICATIONS.

BECAUSE the subject of this paper is a broad one, and its discussion can be extended to an undefined limit, I shall invite attention more particularly to certain points, such as the treatment of the fever, the angina, and the specific complicating nephritis, otitis, and cervical cellulo-adenitis. Others will be cursorily touched upon. It is not anticipated that anything new or startling will be offered you in this article. What I have to say is based upon my clinical observations, made upon nearly one thousand cases of the disease occurring in my own practice during the last fourteen and a half years. These cases naturally have ranged in severity from those of scarcely a week's duration to those which have dragged their length into weary months. The collation and comparison of the clinical experiences of many practitioners ought to be both profitable and instructive. This last-expressed idea is therefore the *motif* of the present essay.

Fever of a higher or lower grade, and pharyngeal irritation more or less severe, are the pathological factors which will be met with in every case of scarlet fever. In every case of the disease, therefore, measures must be adopted to combat these conditions. I will discuss the treatment of the fever first.

It has been quite firmly established that the human organism can be subjected to a high degree of temperature and not succumb. Though this be true, it does not follow that a *laisser faire* policy must be pursued as regards the fever; for in my opinion, if the fever can be reduced by art, the patient is made correspondingly more comfortable, and whatever tends to increase the comfort of the patient assists in directing the attack of illness, as a whole, to a favorable termination. Before the introduction of the recently-discovered antipyretics,

it had been my custom to treat the fever with aconite. The preparation which I employed was the ordinary tincture of the root of the plant of the United States Pharmacopœia. I administered it in doses of one-half drop to one drop, according to the age of the patient, every hour in the beginning of the attack. When the fever had been reduced in consequence of its absorption, I prolonged the interval to every two or three hours, according to the register of the thermometer. Aconite is a remedy "which," as Dr. Charles S. Wood once aptly said in a discussion before the Northwestern Medical and Surgical Society of New York City on the use of this remedy in the treatment of pneumonia, "should go hand in hand with the thermometer." When the child is less than three years old I use the smaller dose, which is increased proportionally with older ones. When the temperature is very high, as 105° or 106°, I have employed with benefit the mode of administration of the remedy so highly recommended by Sidney Ringer, of London,—that is, one drop of the medicine is given every fifteen minutes for one hour, thereafter hourly two or three times, after which the interval is prolonged, and its further administration is to be decided by the thermometric record. Concerning the *modus operandi* of the drug I shall not speak, merely recalling the fact that it is classified as a cardiac sedative. Because of this depressing effect upon the heart, its use has been condemned by some able and celebrated physicians. I, however, have never had any bad results from its use. In the milder cases of the disease, and in cases occurring in robust patients, the system was enabled to tolerate the medication. In cases of an adynamic type, stimulating treatment was also very soon inaugurated, which *pari passu* helped to mitigate, if not to prevent, any deleterious effect of the remedy on the heart's action.

The reduction of the temperature was always quite speedy. Without entering upon a detailed history, I will quote from my case-book the daily temperatures of several cases.

	First Day.	Second Day.	Third Day.	Fourth Day.	Fifth Day.	Sixth Day.
Case I.....	103°	101½°	101°	100°	98¾°	98½°
Case II.....	102°	101½°	100°	98¾°	98½°	98½°
Case III.....	103°	101°	100¼°	100°	99½°	98½°
Case IV.....	101½°	100½°	100°	100°	100°	99½°

Many more could be cited to show the gradual daily decline of the fever under the use of the aconite in the doses mentioned, but I must forbear, lest the tedium of their recital should become wearisome.

When the phenic series of antipyretics were introduced, I abandoned the use of aconite, and employed the former in the treatment of scarlet fever. My experience both with dimethyloxyquinizine (antipyrin) and acetanilide were not so favorable as with aconite. True, the fever was diminished, but my cases did not appear to do so well generally. It seemed to me that the reduction of the fever was accomplished at the expense of the vitality and general well-being of the patient. My patients seemed to suffer more discomfort and prostration, while at the same time the respiratory centres seemed to be unfavorably affected, as evidenced by palpable signs of anxious dyspnœa. Cardiac syncope was also threatened in a number of cases. The doses were not large, averaging from two and a half to five grains. I am not prejudiced against these remedies, for it is my impression that I was the first physician in this country to use antipyrin for fever, and in the first public report here in America on its action, which was made by me on October 15, 1884, before the Northwestern Medical and Surgical Society of New York City, I spoke very enthusiastically about it, though it was derided by the late Professor Austin Flint at that very meeting.* But for the reasons alluded to I soon forsook the treatment with these newer antipyretics, and resumed that with aconite. My later experience with this remedy only corroborated my former observations, and strengthened me in my belief that aconite was the best febrifuge to be employed in the treatment of scarlet fever.

With the hydropathic treatment of scarlet fever I have had no experience, since none of my cases have exhibited extreme hyperpyrexia, and the medicinal agents have always been able to reduce the temperature sufficiently in other cases. In the average case of the disease, however, I would hesitate

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* *New York Medical Record*, vol. xxviii. p. 364, and vol. xxix. p. 648.

very much before applying this method of treatment, because I think that other remedies are preferable. Only when the case had become desperate, and death from hyperpyrexia stared the patient in the face, could I be induced to resort to it.

During the recent epidemic of scarlet fever last winter and spring in New York City, of sixty-three cases of the disease treated by me with aconite, only three died, all of which were caused by severe cerebral complications, terminating fatally within the first three or four days of the attack.

When moderate hyperæmia of the throat exists, very little treatment is necessary, yet it is neither safe nor judicious ever to ignore even a mild angina. Small doses of chlorate of potash, from one-half grain to three grains, proportioned to the years of the child, either dissolved in water or placed dry on the tongue, will suffice. If the anginose condition be more severe, the addition of a few drops of the tincture of chloride of iron to the first-named medicine will be beneficial. If the pharyngeal inflammation be of a severer grade still, larger doses of the martial tincture should be given, even to the extent of twenty drops per dose, in combination with the chlorate of potash.

The chlorate of potash must, however, always be used with circumspection in children, for they are much more susceptible to its toxic action than adults, lest, if too large doses be permitted, serious nephritic lesions may be produced. In my opinion a child should never receive more than three grains at a dose.

If follicular deposits appear in the crypts of the tonsils, I ordinarily prescribe the hyposulphite of soda, in doses varying from two to five grains, dissolved in a teaspoonful of some aromatic water. When extensive exudations of a diphtheritic character are present, I have been induced to use, because of the good results obtained with the remedy in the treatment of diphtheria and diphtheritic croup, the bichloride of mercury; and in the treatment of scarlatinal exudative angina, I must confess that equally satisfactory results have been obtained.

When these remedies seem tardy in their action in the cases in which the throat is severely involved, I have supplemented

their use with local treatment, applied by means of the spray atomizer. For its solvent action I use trypsin in the presence of an alkaline solution, and for its disinfectant and deodorant effect, Lugol's solution in the atomizer, alternately. In all cases accompanied by fetor of the breath I use either the Lugol solution or a solution of carbolic acid as a spray for its correction, whether the case be mild or grave. The treatment of the exterior of the throat will be considered in another connection later in this paper.

In the course of any case, simple as well as severe, of scarlet fever, one must not be astonished to be suddenly confronted with any variety of complication, for the specific cause of the disease is of such virulence that the point of weakest resistance in the patient's organism will most likely be called upon to withstand its assaults. The most usual complications are acute nephritis with dropsical effusions and albuminuria, acute otitis media, and cervical adeno-cellulitis. I have named them in the order of frequency in which they have fallen under my observation.

In the treatment of the renal complication, which has occurred in about six per cent. of my cases, I insist upon the observance of three cardinal principles,—namely, rest in bed, warmth of the surface of the body, and an unrestricted milk diet.

When the urinary excretion is diminished and œdema coexist, I administer a diuretic, preferably the acetate of potash, in the usual doses, combined with the tincture of digitalis. At the same time free catharsis is procured by the administration, at frequent intervals, of small doses of the solution of citrate of magnesia in younger children, and the compound jalap powder in combination with an additional quantity of calomel for older ones. When hæmaturia is present, which I have seen quite frequently, I have sometimes given, with benefit, gallic acid, in three-grain doses, repeated every three hours. At other times five drops of the colorless hydrastis in a teaspoonful of water every three hours has been efficacious to arrest the bleeding. I also order a daily counter-irritation of the region of the kidneys by means of sinapisms. When there is great general anasarca and abdominal ascites, threaten-

to difficulty in its arrest. I met with such an example a few months ago, in which for nearly two hours I was unable to stop the flow of blood.

If by inspection in severe cases we can satisfy ourselves that the tympanum is projecting and is highly congested, presenting the appearance as though pus was accumulating behind it, the membrane should be incised, to liberate the imprisoned matter. This procedure will be followed by a great alleviation of the patient's symptoms. Even if no pus be present, but an intense hyperæmia only, *paracentesis tympani* should also be done, because by the release of the blood the patient's symptoms will greatly improve. When otorrhœa is profuse, whether of offensive odor or not, it is necessary to keep the auditory canal clean and free. In the simpler cases lukewarm saline solutions are indicated, and when fetor is present the addition of a disinfectant and deodorizer, such as boric acid, carbolic acid, Labarraque's solution, or permanganate of potash, in sufficient strength, should be added. I have used all of them at various times. This washing-out of the canal I advise to be done several times daily. After its completion the ear is gently wiped out with absorbent cotton, plain or medicated. Insufflation of the dry powders I do not use, because I think they would interfere with the free drainage and outflow of the discharges, and thus, perhaps, favor a possible extension of the inflammation to deeper structures, and even to the meninges.

After all acute symptoms have passed by I make use of astringent solutions to arrest the residual otorrhœa, usually a solution of the nitrate of silver of five or ten grains to the ounce of water. Of this solution a drachm is added to a pint of lukewarm water, the whole of which quantity is used at one sitting, and the operation is repeated three times daily. After the syringing has been done and the canal wiped dry, I instil in the ear three to five drops of sweet almond-oil, which has been gently warmed in the following way. A teaspoon is dipped in warm water; the drops of oil are poured into the spoon thus warmed, and from it let fall into the ear. I forbid the introduction of cotton pledgets into the ear, but at the same time caution strongly against the exposure of the affected

side to direct currents of cold air. The course of treatment thus conducted has always given me the best results and the greatest satisfaction in the management of aural complications.

In the treatment of disease one can do too much oftener than too little. This reflection applies with great force to the treatment of cervical cellulo-adenitis. When the laity observe the swelling and the œdema on the exterior of the neck, the temptation is to do a great deal and a great variety of things to relieve it. But what is the result? By their rude and energetic, though well-meant, manipulations, the inflamed structures are bruised and further irritated, the inflammatory action is increased, and the very result follows, in consequence of their active maltreatment, which they had hoped thereby to prevent, or at least to dissipate. When there is simple enlargement of the glands of the neck, with but moderate infiltration into the surrounding cellular tissue, I tell my patients to let it be, to do nothing, for nature will probably be able to cause absorption of the diseased products by the time that the attack of illness shall have run its course. Usually my prediction is verified. When the inflammation is of a severer type, I prescribe an ointment composed of slightly carbolized lanoline or vaseline, which is to be gently stroked with the finger-tips, but *not rubbed*, over the swelled tissues. The ointment should be heated a little. I emphasize the injunction that it must not be rubbed, lest by such pressure irritation should be produced, which should be avoided. If, however, appearances indicate that absorption will not take place, and suppuration threatens, then I order hot flax-seed and slippery-elm poultices to encourage it. As soon as fluctuation is palpable a free incision should be made and the pus thoroughly evacuated. The resulting wound and cavity are then irrigated with a phenated solution of the strength of one to a thousand, after which the poultices are continued for a short period. When it is evident that suppuration has terminated, basilicon ointment, impregnated with a few grains of iodoform, is applied to the wound until it shall have healed. The disinfectant irrigations are also repeated at intervals until the final closure of the incision. It is interesting in this connection to refer to the case of a little girl, five years old, who came under

my care with an attack of scarlet fever during April last. She suffered with a bilateral cervical adenitis. On the right side the œdema extended up over the angle and ramus of the jaw, over the cheek and molar bone to the upper eyelid, causing the eye to close. Much to my surprise, suppuration did not occur, but with the gentle treatment outlined above the exudation was by degrees entirely though slowly absorbed. On the other hand, on the opposite side, which was not so highly inflamed, the tissues took on suppurative action, requiring incision for its cure. Though this patient subsequently also developed nephritic and rheumatic complications, she is to-day entirely well, having successfully recovered from the entire train of dangerous lesions. Though it may be a digression, I cannot forbear at this juncture to speak of the most formidable case of cervical adeno-cellulitis which has ever come under my observation. The patient was seen by Dr. J. Lewis Smith and myself in consultation. He was between three and four years old, and had passed through several weeks of an attack of scarlet fever, during which time he had been treated by another physician. We found that he had had an extensive cervical cellulitis on the right side, which had been accompanied by such profuse suppuration and deep ulceration and sloughing, that a cavity large enough to hold an egg had been excavated. At the bottom of the cavity the pulsating carotid artery lay exposed to our gaze, and its coats also were already quite deeply involved in the gangrenous process. We knew of nothing to hinder the fatal issue; ligation would have been a delusion and a snare. Early on the following day the vessel opened, and in a moment death claimed his own. I do not know whether a parallel case is on record, and its rarity has induced me to relate it at some length.

As already intimated, any form of complication may be expected in scarlet fever; among the less rare is acute articular rheumatism, for it is met with quite often. Its treatment does not vary at all from that suitable in cases of the regular idiopathic form of the disease. The salicylates have given the best results in my hands, especially when alternated with alkalies. The tendency of the rheumatism to involve the cardiac muscle and its sac in its embrace is not increased,

because it assumes the *rôle* of a complication of scarlet fever. Certainly, one must be on the alert to forestall as far as possible any such trend of the rheumatic poison by watching the heart, and, on the slightest suspicion that such a contingency is to take place, treatment proper to oppose it must be at once inaugurated.

As regards the chorea, which not infrequently follows this combination of scarlet fever, rheumatism, and heart difficulty, I would state that I have followed up the treatment just alluded to with the administration of arsenic and iron for the choreic condition, and of the bromides, sometimes reinforced with chloral hydrate, to control the excessive nervous excitation.

The most formidable and most fatal complication of scarlet fever which we are called upon to face, is, in my opinion, acute cerebral meningitis, or encephalitis. The symptoms of this complication are in the majority of cases developed early in the course of the disease, and always with great intensity. The prognosis is always much more grave and unfavorable than when the cerebral affection is primary. Whether the aggressive character of the complication is due to a profound saturation of the patient's system with the specific disease, entity of scarlet fever, or to this particular localization of inflammatory action in the course of the fever, I am not prepared to say. True, hyperpyrexia is commonly present in these cases, but I do not think the usually severe course and fatal result are to be attributed to this factor. Treatment of this complication is very unsatisfactory, and I regret to say almost always without avail. Iced applications to the head, the bromides, chloral, and opium in guarded doses, to control the congestion and restlessness, in conjunction with the routine treatment of the fever, constitute the measures which I have employed.

Several cases of acute pleurisy with effusion have also come under my notice as a complication of scarlet fever, independent of serous effusions attending renal disease. They terminated favorably, the effused liquid having been reabsorbed in consequence of a tonic treatment, aided by the exhibition of diuretics, mild cathartics, and counter-irritation to the chest.

I have had very few complications of the eye, indeed, and

none at all of a serious character. In these mild cases the treatment was not modified in any way, but carried out as though the eye affection was a primary disease.

The vomiting, which is usually the initial symptom of scarlet fever, does not call for any special treatment, for it will cease spontaneously as soon as the stomach shall have been emptied of its contents. In a few cases anorexia persisted, but it was finally controlled with ice-pills, bismuth, and lacto-peptine, and carbonated drinks.

Diarrhœa is more apt to complicate the disease than constipation. In a number of cases I have witnessed an almost intractable dysentery, which required very energetic treatment before it was checked. I can recollect one case last winter in which the dysenteric discharges continued for nearly three weeks. In simple diarrhœa the milder astringents and corrigents, as chalk mixture, bismuth, acetate of lead, with paregoric, have always been efficacious. In the graver forms of dysentery, I have obtained the best results from the administration of the elixir of coto bark, in doses ranging from ten or twelve drops to half a drachm in a teaspoonful of water, repeated hourly. When pain and tenesmus are prominent symptoms, I add to the coto from one-half to two drops of Squibb's liquor opii compositus, which is discontinued as soon as those symptoms have disappeared. The opium is given every two or three hours as the urgency of the symptoms indicate. If the patient betray any symptom of narcosis, or undue susceptibility to the action of opium, careful watching is necessary, and it must be abandoned before its dangerous effects should ensue.

For the constipation any of the milder cathartics will do, but what I have used with satisfaction are the suppositories of glycerin, which have been lately brought into use.

Whatever other complications, and to which I have not alluded, may occur, they must be treated in accordance with those principles which their symptoms and location may determine; for, as this paper is founded on my own experiences in the treatment of this disease, I have treated chiefly of those complications which actually fell under my care.

It yet remains to consider certain general principles of

treatment applicable to the management of the disease. Isolation of the infected individual is the only prophylactic treatment of which I know. This quarantine must be an honest one, and must be maintained until the period of desquamation shall have been fully completed. The attendants are also to observe certain obvious restrictions as regards intercourse with the outside world, clothing, and other minutiæ, with which we are all familiar. The sick-room must be properly and sufficiently ventilated, and not so hot as the patient's family are prone to have it for fear that the patient may "take cold." Scrupulous cleanliness about the body-linen and bed-clothing is to be strictly enjoined. The diet should be a liquid and easily-digestible one, suitable to the age and the condition of the patient. If the attack be a mild one, stimulation can be dispensed with; but when it threatens to be severe or prolonged, I do not wait until my patient is debilitated before resorting to stimulants, but I at once begin with them, in order to counteract any such tendencies, and continue their use, until convalescence has been firmly established. I employ alcohol, preferably in the form of whiskey or champagne, and augment its action by the exhibition of digitalis and sparteine sulphate. These two last-named remedies I often combine in the same menstruum, allowing from two to five drops of the tincture of digitalis and from one-sixty-fourth to one-tenth grain of the sulphate of sparteine, for instance, in a drachm of camphor-water every three or four hours. Sometimes, when great prostration is imminent, I prescribe the aromatic spirits of ammonia, in addition to the other cardiac stimulants, in doses of from three to twelve drops in a teaspoonful of water, every fifteen minutes, until the patient shall have revived. In such cases the food is also administered in as concentrated a form as possible.

The ordinary disturbances of the nervous system dependent upon the presence of fever, the common antispasmodics, as the bromides and the like, will control.

To allay the itching of the skin I have at times employed inunctions of feebly-carbolized white vaseline. But it has often been a question with me whether it did not interfere with desquamation and retard the necessary activity of the

skin. I have therefore often substituted for them, when desquamation began, a course of mildly antiseptic tepid baths, either carbolated or sublimated, which seemed to me to hasten the desquamative process, and to produce a healthier action of the skin.

When the eruption is faint or tardy in its appearance, or when it shows a tendency to fade away, in the vernacular "to strike in," I have resorted to warm baths, to which mustard has been added, and to the application of sinapisms to the various portions of the body which appear to be the palest; besides which, ammonia is administered internally. In the majority of cases the eruption then resumes its brilliancy.

The patient must be confined to the house, even in the very mildest type of scarlet fever, and must be protected against all exposure to the vicissitudes of the weather.

The general management of the patient, after having passed through the attack, will, from the nature of the case, consist mainly of tonic and sustaining measures, coupled with a proper observance of hygienic principles.

THE

ETIOLOGY OF EMPYEMA

IN CHILDREN.

BY

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THE ETIOLOGY OF EMPYEMA IN CHILDREN.

I AM certain that you will all agree with me when I say that the subject of the etiology of empyema in childhood is one of the first importance. This theme and its kindred factors are at the present time subjects of the most sincere and painstaking investigation all over the world. The present communication is only a preliminary one to the more complete paper to be published at some future day. In it the special aim is to inquire into the etiology of empyema in infancy and childhood from the stand-point of modern bacteriology. In infancy and childhood the factors of predisposition and exciting cause are modified in their action by the nature of the material dealt with, a growing, developing organism. This is seen particularly well in the chest, where severe processes are borne with a resistance and impunity not to be expected in the adult. We know that in practice these conditions will vary the prognosis. In the disease empyema, it is the etiology which explains in part why the treatment of the disease is more satisfactory in childhood than in the adult. In studying the disease we must revert constantly to the adult subject, and I confess that when these studies were first begun such reference to the adult was obligatory, for none of the works extant treat of the disease in childhood in a satisfactory manner. I found in the course of my work certain similarities between the bacteriological results as published in the adult and in childhood tending to uphold in important points the

dentity in the pathology of the affection as common to the child subject and the adult. A brief *résumé* of the investigations in the field is as follows: The most complete series of observations upon empyemas was published by A. Fraenkel (*Ueber die bakterioscopische Untersuch eiterige Ergüsse Charité Annalen*, 1888, p. 14). He had published fragmentary communications upon the same subject at a much earlier day, anticipating Weichselbaum, who, as early as 1886, appeared with a paper (*Wiener Jahrbücher*) confirming the most important views put forth by Fraenkel. Previous to this, the notices in the literature of bacterioscopic work were imperfect, but they gradually led up to the more complete investigations to be detailed. Flesch and Vaillard (*Archives de Physiol. normale et pathol.*, 1886, p. 162), as also Landouzy, doubt the possibility of a genuine sero-fibrinous pleurisy as the result of exposure to cold. According to these authors all pleurisies whose etiology is not evident are the result of an expression of a localized tuberculosis. The exposure to cold only plays a predisposing *rôle* in the etiology. In sero-fibrinous pleurisies the diagnosis is more difficult because our methods of investigation yield a negative result in most cases. We are thus allowed a mere surmise upon their true character. Ehrlich (*Beiträge zur Ætiol, etc., Pleurit Ergüsse Charité Annalen*, 1882, p. 207) investigated with stainings the bacterial character of fluids obtained from the chest in forty-five cases of pleurisy. Of these, nine cases were tubercular, twenty simple pleurisy, six carcinomatous, and nine empyemas of various kinds. A negative result by cover-glass stain does not exclude tuberculosis. In nine cases the sputa of which showed tubercle bacilli, the bacilli were found in the pleuritic exudate in only two cases. Ehrlich thinks that the bacilli became entrapped in the fibrin masses of the pleuritic exudate, and thus escaped detection in the cover-glass. A. Fraenkel does not reject the possibility of a sero-fibrinous pleurisy as the result of an exposure to cold. In these cases the vital forces are so reduced by cold as to enable micro-organisms situated in the subpleural tissues to become potent. Cold may in this way be an important predisposing factor in the causation of pleurisy. In this class of cases, where micro-organisms have not been isolated, a certain

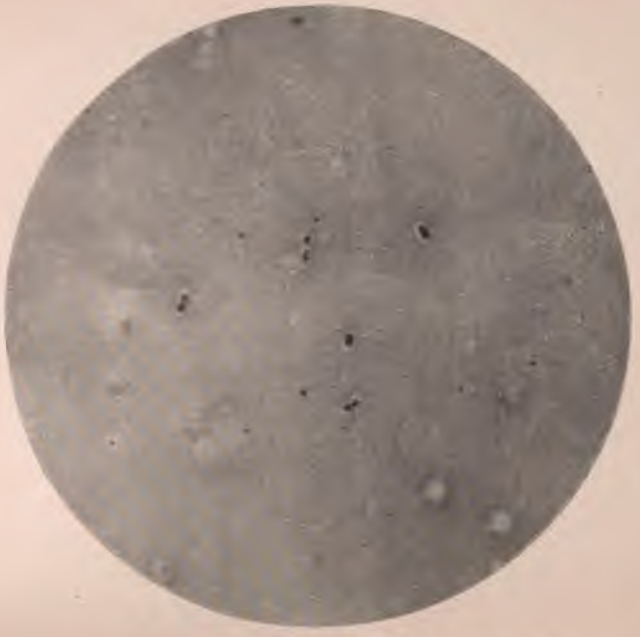
ings made. When pure cultures were obtained from the agar or bouillon in the case of the diplococcus pneumonia, or from agar, gelatin, and bouillon in case of streptococci or staphylococci, they were used for experimental work. Or the pus was, according to Weichselbaum's method, inoculated into agar tubes, and when sufficient growth was attained new tubes and plates were made, the latter to test the purity of cultures. The above was carefully carried out in each case. The media used were blood serum, agar-agar, gelatin, glycerin-agar bouillon, and potato. I found that the most uniform results were obtained with the agar invented by Weichselbaum; it has the requisite consistency and moisture. The pure cultures were suspended in sterile-distilled water and injected into animals, or the pure bouillon culture was injected. Rabbits, guinea-pigs, and rats were the animals used. The cover-glasses which had been prepared with crude pus were stained with methyl violet or with gentian violet by the Gram method. They were stained also for tubercle bacilli. I have determined to report to you my results in twelve cases of empyema, and I will divide them, as Fraenkel, into groups.

Group I.—This group includes those cases in which the bacterioscopic results are not uniform, and in which the micro-organisms found are not diagnostic. The streptococcus pyogenes or the staphylococcus pyogenes aureus were found. I have three such cases to report. Thus far they occurred in children aged seven years (female), eleven months (male), and twelve years (male), respectively. In all of these cases the history, prior to and during illness, failed to elicit any clinical possibility of tuberculosis. In all, the bacterioscopic results were as above. I leave the clinical histories of all the cases of this paper for future publication. In two cases the streptococcus or chaincoccus was found which in every way as to growth and action corresponds to the streptococcus pyogenes. In the third case, a child, aged eleven months, the staphylococcus was found which in every way corresponds to the staphylococcus pyogenes aureus. The above group finds its exact counterpart in the group of similar cases investigated by Fraenkel in the adult. Eliminating the presence of tuberculosis, we are still in doubt as to the exact etiology of these cases; we might with

Fraenkel suppose that they could have complicated a pneumonia, for Weichselbaum proved that in pneumonia there were present (as mixed infections) both the streptococcus pyogenes and the staphylococcus pyogenes aureus. Again, these empyemas could arise spontaneously, as explained above, with the predisposing aid of exposure and cold or a slight traumatism, the above micro-organisms being present in the subpleural tissues. In none of my cases was a traumatism mentioned in the history. On the whole, it seems uncertain as to how these empyemas originate. The organisms found, as you see, are not characteristic.

GROUP II.—This group includes those cases in which I have been able to establish the presence of the pneumococcus of Fraenkel and Weichselbaum in the purulent exudate. This organism was found in pure form and uncontaminated in all the cases of this group. I was perhaps fortunate in working with a simple purulent exudate and obtaining my specimen pure and uncontaminated. In all the cases the crude pus spread upon cover-glasses and stained showed the capsulecoccus singly or in pairs or chains, and the lancet-shapes could be beautifully seen. These diplococci could be well stained by the Gram method. The pure cultures with Weichselbaum agar were characteristic, and I leave their description for a more lengthy paper. There are seven cases to report in this group, six males and one female child, aged five years, twelve months, two and a half years, three and a half years, two years, and two and a quarter years, respectively. I wish to direct particular notice to two cases of this group. In both of these cases the effusion, when drawn from the chest with the hypodermic syringe, was at first clear and serous, in one case being devoid even of fibrin flocculi. In both cases, however, the diplococcus pneumonia was isolated in pure culture, and its virulence established upon animals. This micro-organism could also be stained by spreading a drop of the crude exudate upon cover-glasses. If a drop of this exudate were placed under the microscope, the diplococcus could be seen, as also a few lymphoid cells. These cases subsequently became pronounced empyemas, the serous having turned into a purulent exudate. This tends to support the assertion of Fraenkel that when an

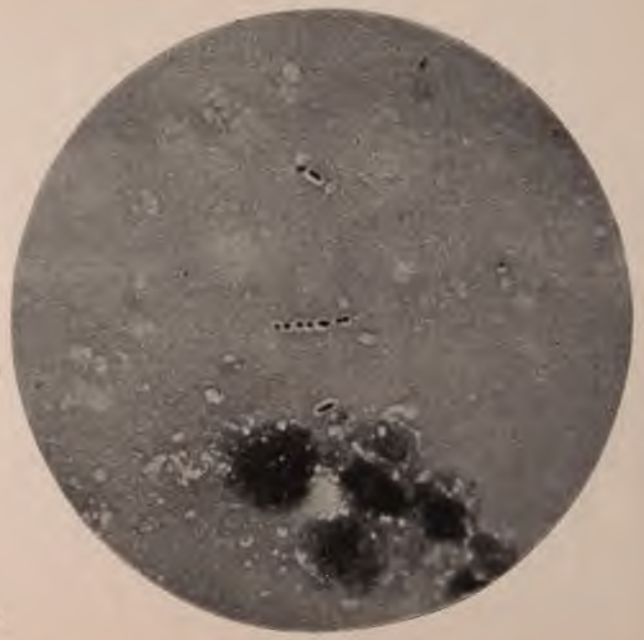
PLATE I.



(MICRO-PHOTOGRAPH.)

1. Diplococcus pneumoniæ in the crude pus of Group II. spread upon a cover-glass and stained with gentian violet; shows the capsule; Zeiss apochromatic lens, projection ocular No. 2; × 1000; zircon light.

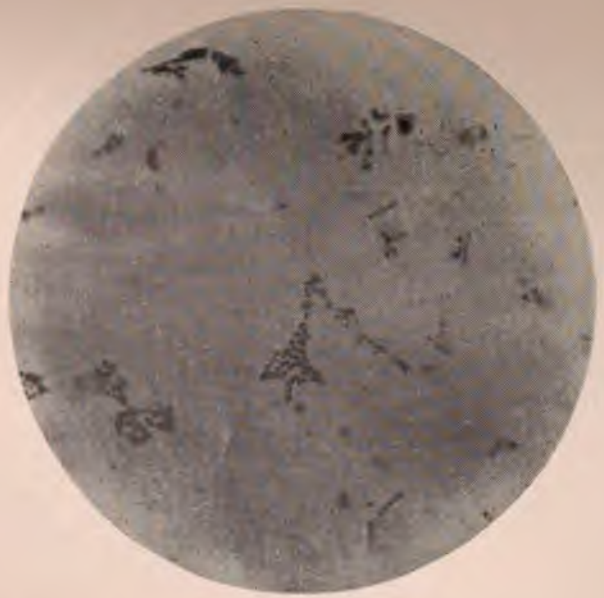
PLATE II.



(MICRO-PHOTOGRAPH.)

2. Diplococci pneumoniæ in the crude pus of empyemas of Group II. Shows capsule, diplococci single and in chains, also the "candle-flame shape." Cover-glass specimen; gentian violet; Zeiss apochromatic lens, projection ocular No. 2; × 1000; zircon light.

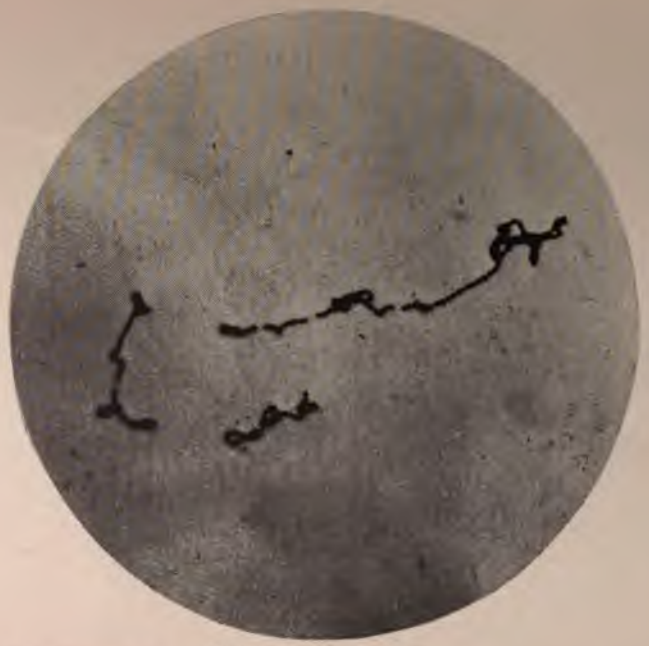
PLATE III.



(MICRO-PHOTOGRAPH.)

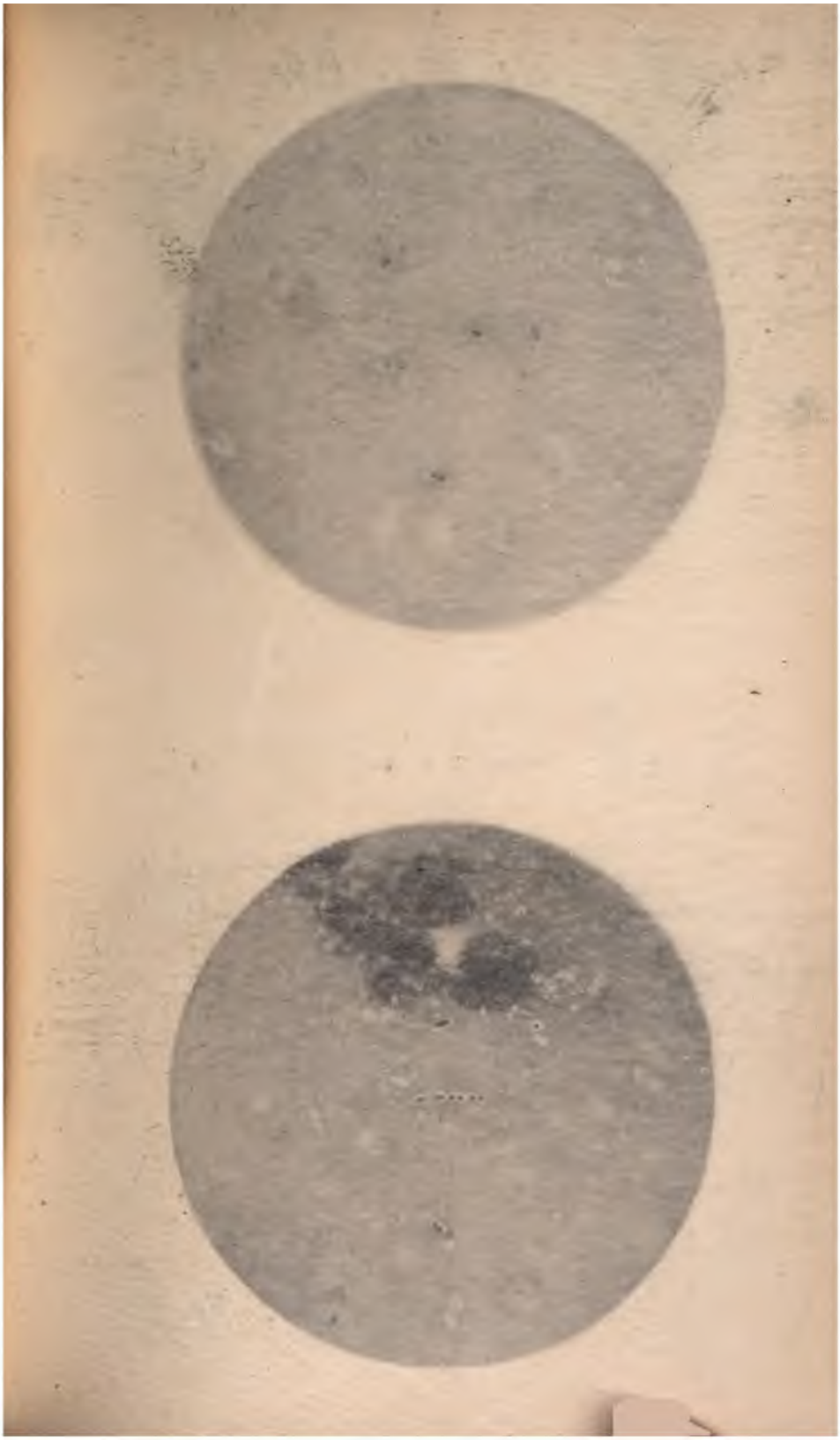
3. Pure culture of diplococcus pneumoniæ obtained from the pus of a case of empyema in Group II. Agar culture: Gram stain; Zeiss apochromatic lens, projection ocular No. 4; × 1000; zircon light.

PLATE IV.



(MICRO-PHOTOGRAPH.)

4. Pure culture of streptococcus pyogenes obtained from the pus of a case of empyema in Group I. Bouillon culture; cover-glass specimen; gentian violet stain; Zeiss lens as previous specimen; × 1000; zircon light.



exudate, serous in character, contains streptococci, it can be predicted that it will shortly become markedly purulent. The same could be stated of the diplococcus pneumoniæ, for we know of its established capabilities to produce suppuration. The above also shows that the character of a serous exudate cannot be established until examined bacteriologically. In this way we can make a differential diagnosis between those exudates which, like the above, are acute in character and those which are serous, and contain no micro-organisms and fail to give any results with our media, and the majority of which, in the *adult* at least, are tubercular. The presence of a serous fluid in the hypodermic may be of little use in proving the purulent character of the fluid in the chest as far as gross appearance is concerned. It is well known that the pus of empyema, even in the chest, has a tendency to separate into two portions; that portion in the dependent part of the chest may contain most of the lymphoid cells and be markedly purulent to the macroscopic examination, while the upper stratum which we have aspirated may be serous. The clinical diagnosis of pleurisy with serous effusion is only temporary at best, and we must be always on the lookout for pus, especially if the serous fluid aspirated contains streptococci or diplococci. It has been customary formerly to assume that if a fluid has been serous at the first aspiration and subsequently became purulent, it had been contaminated from without by the hypodermic. If such effusions had been carefully examined, I am sure micro-organisms would have been established at the very outset. When a serous exudate has from the very first been proven free from organisms and then subsequently turns purulent, then we may think of some contamination or mixed infection. When the bacterioscopic result is as positive as in the cases of this group, I think it is not necessary to prove the clinical presence of a pneumonia in order to trace the origin of the empyema. The isolated presence of such a virulent micro-organism as the diplococcus is sufficient proof as to the origin of such inflammatory processes of the pleura. As stated above, the cultures of all the cases in this group were inoculated upon animals with results identical with those of Fraenkel and Weichselbaum. I shall publish them in the near future.

GROUP III.—Empyemas occurring in tubercular subjects, as far as investigations in the adult teach us, may be encapsuled and localized to narrow limits by adhesions in the chest, or they may be diffused over the whole pleural surface. They may complicate a tubercular condition of the lungs, or they may be present when there is no pulmonary tuberculosis. The general result is not changed by these conditions, the prognosis is always bad and conditional: a partial recovery is the result in the most favorable cases. The tubercular cases in the adult are the most unsatisfactory to treat. Though we relieve certain symptoms, other conditions are superimposed by our treatment which are not less harassing to the patient than were the symptoms of the original condition. In adults the bacteriological investigations may (Fraenkel) yield an absolutely negative result. Our inoculations of the pus from the pleural cavity upon various media yields us nothing, and thus far experiments upon animals have been equally unsuccessful. This may be because our present methods and media fail to reach these cases; or, better still, because, though the tubercle bacilli may exist in the pleural adhesions and the tubercle tissue of the adhesions and thickened pleura, they may be present in such small numbers in the exudate, at least in that withdrawn from the chest, as to fail to grow when artificially cultivated. When a case, upon careful examination of the pus, gives an absolutely negative result, we are justified in assuming the possibility of a tuberculosis. In most of these cases where post-mortem has been made tuberculosis has been established. The second class of cases of a tubercular nature are those in which the sputa of the patient and the pus of the empyema show tubercle bacilli, both by stain and culture. Lastly, streptococci have in some cases been found to contaminate the pus in some of these tubercular cases (Fraenkel). In this last set of cases a contamination is assumed from the subpleural tissues or the lung. In children, as far as I know, no parallel investigations to the above are on record. I have only one case to report, a boy of eight years, in whom the empyema is tubercular. In this case tubercle bacilli were found in the pus of the chest, but it was contaminated by the presence of streptococcus pyogenes.

This corresponds to an adult case of Fraenkel's. This patient is still alive, and, though walking about, carries a large suppurating cavity in the chest, which refuses to close in spite of the enormous contraction of the chest wall and extensive resection of numerous ribs. There are no signs of tuberculosis discoverable in the healthy lung. It was not possible to obtain the sputa for examination. Thus far my experiments upon animals with the pus have been negative, the results being clouded by the presence of streptococci. It is not at all impossible for a tubercular empyema to make, if localized, an apparent temporary recovery; but whether such recoveries become permanent it is at present impossible to say. It is also problematic in these cases as to what is the real focus of trouble; why should a tubercular area in the lung, for a long time dormant, suddenly cause an empyema? We could here also invoke the predisposing exposure or traumatism. Tubercular empyemas are the most difficult to diagnosticate in some instances during the life of the patient. In children it is not always possible to establish a tuberculosis of the lung by physical exploration; the sputa is also difficult to obtain. These little patients have been so harrowed by the disease and its results, and the presence of paraphernalia of the ward is so associated in their minds with suffering, that when a bottle or spit-cup is placed before them they resist all attempts to obtain a specimen of sputa.

Group IV.—The last group of empyemas in children belongs to that class of cases in which we can, with a degree of probability, point to a focus of suppuration situated outside the chest as the possible source of infection. These cases include pyæmic infections, and correspond to that variety of empyemas which, in the adult, are found complicating puerperal cases, or cases with retropharyngeal abscess or peritonitis (Fraenkel). The case which I desire to place in this group occurred in a child aged four months, breast-fed, which, for two weeks previous to the chest-trouble, had been suffering from a deep burrowing abscess on one foot. Just before the appearance of this abscess the infant had been vaccinated, and at the time of consultation for the chest-trouble the vaccine pustule looked quite angry. The pus, withdrawn with the

usual precautions from the chest in this case, contained streptococcus pyogenes, which, upon inoculation, proved virulent in action. An animal inoculated with this pure culture died with multiple abscess of the liver and lung, general jaundice, and a large soft spleen. This case proved fatal in two days after pus was established in the chest, in spite of operation, dying with all symptoms of pyæmic infection. No autopsy was permitted.

The conclusion to be derived from the above is a forcible one, tending to show what a large proportion of empyemas in children follow or complicate processes in the lung of an acute character. With early and efficient treatment we can look all these cases hopefully in the face. Even those whose etiology is uncertain do not hold out such a bad prognosis to the patient. The tubercular and pyæmic cases are the stumbling stones of pediatric practice.

DISCUSSION.

Dr. Booker, of Baltimore.—During the last six or eight weeks we have had an unusually large number of cases of empyema among the children at the Johns Hopkins Dispensary, having had five cases within that time, and Dr. Shaw has been making some investigations from a bacteriological stand-point.

The president then called on Dr. Shaw, of Baltimore, who said,—

I may state some of the results of the investigation, as they bear a close relation to the admirable paper just read. It is my regret to say that no examination was made of the pus of the first case. In the remaining four the pneumococcus was found on each occasion. The pus from two of these was inoculated in each case into a rabbit and a mouse, with the results that the pneumococcus was obtained in a pure culture. The mouse of the first case died on the fourth day, and the autopsy showed all of the organs to be infected with the micro-organism as also the blood, but the results on the rabbit were frustrated, owing to the fact that the needle of the syringe perforated the intestine from which a peritonitis was set up, in the fluid of which the colon bacterium was found. I may also say that the pneumococcus was likewise obtained, but no value was attached to the experiment.

In the second series of inoculation the rabbit died on the

third day, with marked evidence of infection in all of the viscera as before, and the pneumococcus was again obtained in pure culture. The mouse lived for some time after, so no value was attached to the autopsy.

In the pus of all examined the micro-organism was in a form we have never seen it before, being in chains of from four to seven in number, and surrounded by a continuous capsule. Some of these had peculiar hooked ends. I might say that it was not obtained in this form from the animals inoculated.

SCORBUTUS IN INFANTS—AMERICAN CASES.

(ILLUSTRATED.)

BY WILLIAM P. NORTHRUP, M.D.,
PATHOLOGIST TO THE N. Y. FOUNDLING HOSPITAL.

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SCORBUTUS IN INFANTS; AMERICAN CASES.*

(ILLUSTRATED.)

BY WILLIAM P. NORTHRUP, M.D.,

Pathologist to the New York Foundling Hospital.

THE object of this paper is to present some cases of infantile scurvy occurring in this country, and to intimate something regarding their frequency of occurrence. As to the essential nature of the disease there is no positive recent contribution to literature, nor has the writer anything new to offer. After the valuable papers on scurvy in children to be found in *Keating's Cyclopædia*, and in that most admirable book of Eustace Smith's, there remains nothing to say until some investigator shall announce the long hidden secret of the essential causative factor which gives rise to the lesions which we, by common consent, denominate scurvy—be it in infants or adults.

The first case reported in this paper is so typical in lesion, course and symptomatology; in its ætiology so suggestive and in its treatment so successful; in short, so typical and instructive is the case, that it has seemed best to the writer to set it forth in detail, with the statement that it illustrates nearly every point mentioned in the two articles referred to above. As these volumes are probably in the library of every member of this section, it is

* Read before the American Pediatric Society, Washington, D. C., Sept. 24, 1891.

useless to duplicate words. If it shall appear that scurvy in children is not of frequent occurrence, there is yet a certain advantage in considering such cases, especially if they are liable to spring up in the path of the general practitioner at any turning.*

CASE I.—*Typical; scurvy occurring in rich surroundings; recovery; patient aged sixteen months.* In the midst of a pouring rain on a Sunday morning, a carriage drew up to the curb, the bell gave two quick jumps and in a few minutes a man was marching up and down the office floor. The cause of his nervousness was quickly announced. His second child, an infant, had rheumatism and had become no better during the fortnight's sojourn of himself and wife in a neighboring capital. The child in their absence had been in the care of an old nurse, so gifted in the art of nursing and, I may add, in self-conceit. On his return home the previous evening, the infant was not well. It was not very sick, so he thought, when he went away, and by this time he supposed it would have entirely recovered—instead, it was worse. He sent for the doctor and, in his anxiety, desired an immediate consultation. His family physician was among the ablest practitioners in New York; he had not been called to the child within a week; had not been informed by the nurse, if indeed he had not been misled by her. The history of many cases can be written backwards better than consecutively, and the present one is of this character. Suffice it to say, in preface, the diagnosis was rheumatism and the treat-

**Scurvy*, by Thos. Barlow. Cyclopædia of the Diseases of Children, Keating, vol. ii., page 277.

CONCLUSIONS—(CHARACTERISTICS.)

"(1). Predominance of lower-limb affection; (*a*) immobility; (*b*) excessive tenderness; (*c*) general swelling of lower limbs; (*d*) skin shiny and tense but seldom pitting and not characterized by undue local heat; (*e*) on subsidence, revealing a deep thickening of the shafts; (*f*) liability to fracture near epiphyses.

"(2) Swelling of the gums, varying from definite sponginess down to a vanishing point of minute transient ecchymosis. . . . .

But to them must be added as:

"(3). The most diagnostic of all, definite and rapid amelioration by antiscorbutic regimen."

ment had embraced salicylic acid and tincture of iron, without success.

At the time of the consultation the father and mother of the little patient were present; both were within the thirties, healthy and vigorous, the father looking like a hardy yachtsman. The family history on both sides was good. The family were luxuriantly housed in the most hygienic surroundings of up-town New York. In short, the infant, whose history we are going to consider, who was suffering from malnutrition, from "insufficient and poor food," to quote words so often used in connection with children's diseases, was the offspring of wedded representatives of two rich and powerful families in the fashionable and political world of the great metropolis. There was no lack of love and devotion on the part of parents or grandparents. The lack was in wisdom and judgment, the abundance, the nurse's self-esteem and ignorance; these allowed the present case and history to continue on to full accomplishment.

The child at this time was sixteen months old; was the second born; a female; thriving very well in the early months of life; at the fourth month the mother's milk failed to be of sufficient quantity and quickly, thereafter, ceased altogether. The first born is still living and robust; the mother's milk had after a few months been insufficient for it, but the child continued to thrive. After the failure of the mother's milk, in our patient's case, one of the proprietary foods was given her. By some misunderstanding this food was diluted with water and milk, the proportion of the latter being too small. For a time the child thrived very well, apparently, though it was rather backward about walking and talking. Its digestion was good, its bowels reasonably satisfactory and it seemed satisfied with its food. It never gave any evidence of rickets; teeth in usual quantity made their appearance at the usual time.

Three weeks before the visit spoken of (this fact was subsequently elicited after questioning both parties) the nurse had noticed some *change in the child's gums*,

After reflecting a week upon this she called the attention of the mother to the same. The change was not marked. While on a visit to another patient in the household, the doctor's attention was called to the child as he was hurrying out to his carriage. The baby was reported as not quite well and nothing was said concerning the gums. He hastily looked her over, learned of the misadventure with the proprietary food, added the requisite amount of milk to bring it up to the prescribed standard, ordered beef-broth, iron peptonate, and was gone.

One week later than this, the patient developed *trouble in the right lower limb*, evidenced by worrying, sensitiveness on handling and tendency to keep the limb nearly straight. Anti-rheumatic remedies were prescribed. There seemed no reason why the case should not speedily come out of its condition of slight depression, now that the food was improved and treatment instituted.

During the succeeding week very little is known concerning the child; the parents were absent from home; the family physician was not called; the nurse drew no conclusions from the, now rapidly-changing gums, and as to the rheumatism, the progress was slow. It is not difficult for physicians to understand how this infant, once started on its career of malnutrition, could even in the house of luxury, under these circumstances, develop ripe symptoms. At this juncture the parents returned home to find the rheumatism worse, instead of cured, and a hasty muster of physicians was the result.

The child cried on seeing a strange face, becoming alarmed also for the safety of its lame leg. In the wry face of crying, the little patient fairly *unbuttoned* from between its lips two rows of irregularly nodulated, purplish gums, from the summits of which the points of its teeth fairly protruded. *In the upper spongy row was a depression with ulcerated walls and sloughing shreds. The gums were dark and bled freely* in the act of crying, from compression of the lips alone. There was nothing further abnormal about the face beyond a worried expression; no ecchymoses; no

petechiæ; conjunctivæ were normal; no evidence of unhealthly condition of the mucous membrane of the nose. There was no history of nose-bleeding, no hæmaturia, no hæmorrages from the bowels. The child was now stripped of all clothing and laid on its back on the bed. It continued to whimper, throw its arms about freely, draw up its left leg; as for its right, it could move it slowly, but only a little, and could not be induced to flex it. The *right thigh was somewhat larger than the left*, to observation; by measurement it showed a difference of about two and a half inches, which, considering the thin thighs of the small patient, augured a marked difference. The enlargement was fusiform, greatest just above the knee. Apart from the *spongy gums and swollen thigh* there were no external manifestations.

The diagnosis of this case is scorbutus; what confirmation of this diagnosis can be offered? The answer is, the success in treatment.

The child was removed at once to the country, its proprietary mixture was stopped and in its place were given fresh cow's milk, expressed juice of beef, baked potatoes, also citrate of iron with excess of citric acid. The one thing which this scurvy case seemed to crave, for which it reached out, which it seized with ravenous avidity, was *orange*. The child could hardly be restrained till it held the fruit in its grasp; and then proceeded to souse its lips and nose in the juice. Improvement began at once; in five days its gums were markedly better, in ten entirely normal. As for its thigh, at the end of ten days the improvement was marked both in sensitiveness and size, and a month later the child was standing on its feet. A slight thickening over the femur could be detected for a few days, at the end of which time, the patient seemed absolutely well. "Now," to quote the words of the latest report, "she would strike you as a child small for her age simply, nothing having ever been the matter with her."

Several points may be learned by reading between the lines in this case. As Dr. Charles McBurney once

very aptly said, speaking of the value of original investigation: "There is also great value in *re*-search." At least we may argue, that in cases of spongy gums and pains in the knees it is well to consider the diagnosis of scurvy. A certain amount of sinister interest attaches to the fact that this child was vaccinated a month before the first symptoms appeared, and all medical men connected with the case were catechised as to the near or remote possibility of its causal relation to the lesion of the gums and thighs. It was, by the way, vaccinated on the leg. Needless to add, its course was perfectly typical for an effective inoculation and was followed by speedy healing.

To entirely complete the disease-picture given us by the English authorities, nothing is required but the post-mortem findings, which the following cases furnish.

CASE II.—*Fatal scurvy in a child of eighteen months; Autopsy.**

This child was an inmate of the New York Foundling Hospital and was what is called a "nurse-baby," that is, she was nursed by a mother who, in addition to her own baby, nursed a second of about equal age. Her own child thrived; the second furnishes this example of malnutrition and the pathological specimen which is here shown. The nurse was said to be very fond of the child and "took on" when it sickened and died. Since we are considering a case of scurvy developing in a breast-fed (*sic*) child it is well to bear in mind the above facts and the added fact that nearly all babies nursing two at one woman require to be fed more or less. We are justified in forming our own conclusions as to which was fed more and which less; we know which baby was hers and which was not, which thrived and which developed fatal scurvy; we also have our notions of the tendencies of maternal instincts. However, the history must be recorded as

* Reported in Proceedings of the N. Y. Pathological Society, 1889, pp. 66–67.

those in charge of the case furnish it, and it reads, "the child was wet-nursed the first sixteen months of its life."

Briefly the history of the illness was as follows: This female child when sixteen months old was observed to be failing, and as the history reads, "on account of impaired nutrition was taken from the breast and was given vegetable acids." In the seventeenth month of life, which was one month before death, the right leg and knee became swollen and tender. Temperature was 101° F. After two days the symptoms seemed temporarily to disappear. Two weeks before death and six weeks since the weaning, the child appeared to be very sick, her gums were swollen, smoky-black and bled freely; two days later her left eyelid became swollen, black, having the appearance of the classical "black eye." Temperature thus far continued about 101° F. One week later there developed the physical and rational signs of pneumonia. At this time her other eyelid became ecchymotic and the other thigh markedly swollen.

During the remaining days of life the small patient became excessively anæmic, having a metallic pallor, which gave a particularly wretched appearance with the contrasting ecchymoses about the eyes. Her passages were black and pasty; no petechiæ; the child failed rapidly and died with pronounced symptoms of pneumonia.

Autopsy.—The main interest lies in the condition of the legs. As regards the organs, it is sufficient to say there were no hæmorrhages; extensive pleuro-pneumonia of left side. Left thigh symmetrically enlarged, larger than the right, though both were obviously above normal in size. Left femur was, at its upper extremity, normal, epiphysis and end of shaft. The lower half was invested about with a black grumous subperiosteal layer of blood having a thickness of two to three millimeters. The lower epiphysis was detached, the lower end of the shaft macerated, eroded and soft, lying loose in the

black disintegrating blood-clot. The femur of the companion leg was surrounded for its lower two-thirds by a thinner, black, subperiosteal blood-layer—(See illustration).

The lower epiphysis was not detached though both it and the shaft were congested. No hæmorrhage into any joints. The right and left tibiæ were surrounded by a thin, dark hæmorrhagic layer beneath the periosteum and the proximal portions of both were congested. The fibulæ, likewise the bones of the upper extremities, were normal.

The accompanying illustration is drawn from a specimen which consists of a lateral half of the lower limb of the side less affected.

Microscopical examination of the bone disclosed no syphilitic or rhachitic changes, and no inflammatory changes in bone or periosteum. The softened, macerated bone gave no evidence of suppuration; moderate congestion of the fellow femur and the upper extremities of the tibæ.

CASE III.—Dr. Richard Van Santvoord has reported* the autopsy of a helpless idiot, six years of age, in whom extensive hæmorrhage, with separation of both upper epiphyses, had occurred under the periosteum of the upper halves of both humeri. This hæmorrhage was apparently the immediate cause of death. The case was regarded by him at the time as probably traumatic. Reports that he had subsequently seen of extensive subperiosteal hæmorrhage, with epiphyseal separation, had been regarded by their recorders as scorbutic. He had become convinced that his own case belonged under that heading. Gums were not mentioned as being involved.

CASE IV.—Dr. August Caillé contributes for use in this paper, an *autopsy* record of a typical case of scurvy—autopsy by himself. He says:—"In addition to the external appearances there were subcutaneous effusions of

*Ibid.

[Drawn from Specimen by H. McDONALD, M.D.]

Specimen from a case of Infantile Scurvy, showing subperitoneal hæmorrhage about femur and tibia of the side less affected.

blood, hæmorrhagic spots on divers mucous and serous membranes, swellings at the joints and subperiosteal effusions of blood at the epiphyses."

The three last narrated cases furnish the only autopsies the writer has been able to collect. Bearing in mind the complete picture of disease furnished by the previous cases, let us consider the following ;

CASE V.—Dr. Chas. H. Richardson of the North Eastern Dispensary—children's class—has furnished the following most interesting and illustrative case.

Anne K., aged twelve months, was brought to the dispensary by its mother with the history of "failing" for nearly two months. The child was healthy for some time after birth, when the mother was obliged to work, and of necessity the baby was "farmed out." She reported the food as having been condensed milk, adding the somewhat ambiguous item, "and what was going."

The child had recently vomited blood, passed blood from the bowels and presented petechial spots of subcutaneous hæmorrhage. The gums were slightly softened and according to the mother's account the inside of the mouth had been black. Both legs were enlarged to considerably more than double their normal size. Epiphyseal ends showed marked enlargement. Limbs very tender, hard and pitted slightly on pressure."

"Head sweats. Temp., 100⅘°. Tendency to spinal curvature in dorsal region."

To continue the verbatim quotation of the doctor's letter :—"I considered this a combination of scurvy and rickets and put the child on a live diet of fresh milk, orange and lemon juice." Three days from the first observations and prescription the purpuric spots had mostly disappeared, and no new ones had occurred. The child seemed slightly better ; there was no vomiting, yet the condition of the limbs was unchanged and the temperature continued slightly elevated as before.

The following paragraph gives the case an added interest and accuracy : "The swelling of the limb being due to

subperiosteal hæmorrhage, added to subcutaneous œdema, I wished to see the character of the effusion next the bone. I thereupon passed an aspirating needle down to the femur and drew off some of the effused fluid. It consisted apparently of pure blood, which microscopically showed the red globules, somewhat ragged and disintegrated, and the white ones somewhat increased in number."

At this point the case escaped from observation and the history ends.

CASE VI.—Dr. Hermann Goldenberg, at his class in the Mt. Sinai Hospital Dispensary, observed the following case of scurvy:

A boy, two years old, was brought in by his father, who stated that the patient had been well up to a few days previous, when he noticed red spots over all his body, swelling of the legs, bleeding from the mouth, bowels, nose; also hæmaturia. The food of the child consisted exclusively of milk.

The family history was good; a number of older children were strong and healthy; no malaria; no syphilis. To quote Dr. Goldenberg literally:—"I found a hæmorrhagic eruption in nodes, spots and large patches, up to the size of a palm of a hand, on face, trunk and extremities. The hæmorrhagic character was clearly demonstrated by the fact that the redness remained on pressure, and the changes in color, within a few days. The gums had the characteristic color and form, as in scurvy. There were gangrenous spots in pharynx and on tonsils, swelling of the feet and legs and of the scrotum, through extravasations into subcutaneous tissue. Fœtor of the mouth; urine which the father brought was bloody; general malaise. No signs of rickets." Dr. Goldenberg presented this case before the Mt. Sinai Alumni Association as a characteristic case of scurvy. It was accepted as such. One member temporarily dissented, preferring to call it hæmorrhagic urticaria. The patient was on the following day sent to Dr. Jacobi, who confirmed the diagnosis and pronounced the case scorbutus. Lemon juice, fresh vegetables, potassium chlorate, internally and

as gargle, were ordered and within a few days the child was well.

CASE VII.—The following case was narrated to the writer by the physician to the child, whose case was first narrated. The patient was a boy sixteen months old; the fifth child of a father, of high rank in the army, having a physique which the doctor fittingly typified as by the word "bull"; the mother was a delicate woman; its four or five brothers and sisters were all delicate. The child was well housed and cared for, or to use the words of the narrator, "over-cared for"; was wetnursed for a few days only. Thereafter, it was fed on a proprietary food. Up to the sixteenth month of life it seemed to be well nourished and attain good development. At last, its gums became spongy, then its right ankle became swollen, discolored, bluish, tender, fluctuating. This patient was seen by an able consultant, an honored member of this society, and pronounced rickets—probably meaning *acute rickets*.

On a treatment of lime, muriatic acid and tincture of iron the child slowly and gradually recovered. The symptoms in this case were spongy gums and hæmorrhages about one ankle. No mention is made of petechiæ.

CASE VIII.—Dr. Delafield furnishes the following interesting case. In the month of June, a child about three years old, suffering from obstinate diarrhœa, was brought to him for consultation. He prescribed an exclusive meat diet and advised the parents to take it at once into the country. They did as directed, and the child speedily recovered. So pleased were the parents with the prompt effect of the diet in the pronounced improvement of the patient that they believed they had learned a valuable secret for securing the continued health of the child.

They, without hesitation, settled down to a rigid *regime* of exclusive meat food. This they followed conscientiously for two or three months. In the last of September, the condition of the child induced them to return to town and again consult Dr. Delafield. This time the pa-

tient presented a characteristic case of scorbutus. To use the words of the narrator: "The diagnosis was perfectly clear; had characteristic signs of scurvy." On a rational diet all symptoms quickly disappeared and the child became quite well.

CASE IX.—A letter from Dr. L. Emmett Holt gives the following: "Some points regarding a scorbutus case; the first symptoms appeared in the latter part of April, the infant being at that time eleven months old, and having had all the symptoms of extreme marasmus for several months. From the latter part of February she had been upon an exclusive diet of a proprietary food. There was first noticed a swelling of left knee resembling in general appearance that due to articular ostitis; a week or more later the gums were spongy, purplish, and slight hæmorrhages occurred from time to time. The swelling about the knee increased steadily during the next six weeks until it was fully four inches in diameter; the swelling extended up to the middle of the thigh; there was extreme tenderness and later a slight purplish discoloration of the skin. Exploratory puncture gave blood only. The child died on June 23d. The above symptoms continued together with those of extreme anæmia and exhaustion; there was a slight fever for the last two months; at no time was there any ecchymoses beneath the skin. At the autopsy the swelling of the limb was found to be due to subperiosteal hæmorrhages, the bone being surrounded by a mass of old blood-clots; the periosteum was extensively stripped up; there was complete separation of the lower epiphysis of the femur.

CASE X.—Dr. W. F. Lockwood, of Baltimore, furnishes the following: we quote the letter in full, verbatim.

"Aug. 12th.—Mrs. W——, whose family had been in the country during the warm weather, asked me to visit her child, thirteen months old. She stated that it had been ailing for some weeks with diarrhœa varying in severity, but never entirely relieved. Of late she had thought the child suffered pain in its lower limbs, that it screamed in an unusual way at night, especially when moved or

touched, and that there was increased fretfulness during the day. She referred also to some spots 'like old bruises' on its legs below the knees, more marked on the right.

"It had been nursed until six months old, after which time it had taken almost exclusively a proprietary food.

"Examination showed the child fairly well nourished, rather anæmic, complexion sallow. No evidence of rickets, no swelling along shaft, or enlargement at ends of bones. Ill defined ecchymotic spot on left leg, but plainer on the right. The right leg semi-flexed and everted. No tenderness manifested on gentle manipulation of body or limbs. Gums showed dusky purplish fold at root of each tooth. They had bled occasionally for some weeks and the stool had pretty constantly been streaked with blood. Pulse and temperature were normal.

The diagnosis was made of scorbutus and directions were given to change the diet to fresh milk undiluted, potato and orange or peach juice. A few days after my visit the mother reported the child improved in every way. The diarrhœa had stopped and there was no screaming or restlessness at night. A second visit was not required and the improvement, I have heard, has continued."

CASE XI.—Dr. G. H. Whitcomb, of Greenwich, N. Y., has, since the writing of this paper, published a most illustrative case [*Archives of Pediatrics*, Oct. 1891, p. 760, of *Artificial Baby Food and Scorbutus*.] The child was eleven months old; after fifth month was fed exclusively on artificial food, during which time it grew very fat, "seemed to thrive famously." "When a little over ten months old she became petulant, and evinced a disinclination to move or be handled. The legs were partially flexed and remained rigid. Any attempt to straighten them elicited screams. The gums were spongy and bled frequently; muscular pains were so severe as to deprive the child of rest." "The physician at that time in charge diagnosticated and treated rheumatism. . . . After ten days the family went to Rome, . . . where the diagnosis of rheu-

matism was approved and alkaline treatment pursued for two weeks, after which they came to Greenwich."

In Dr. Whitcomb's graphic account occur such expressions as the following : " screamed when handled . . . limbs resembled Bologna sausages . . . gums were spongy, ecchymotic blebs discharging sero-sanguinolent fluid . . . seven teeth . . . *no evidence of rachitis*." Treatment comprised fresh milk, rare-broiled beefsteak and sweet oranges.

In three weeks the child was restored to complete health. The doctor believes the scorbutus to have been caused by the exclusive use of the prepared food and he concludes that "no cereal or chemically prepared food can nourish perfectly," and should be supplemented with fresh milk, meat and fruit juices.

During the summer just past, the writer has had opportunity in three great medical centres, personally, to compare notes on the subject of scorbutus in young children.

Prof. Ranke, of Munich, said that with him it was "in the highest degree of rare occurrence." He could not at once recall a case.

The assistant of Prof. Widerhofer in Vienna, Dr. Karl Foltanek, at that moment surrounded by a throng of wretched out-patients at the children's hospital, declared that in his experience, and it must be considered a colossal experience, "scorbut" in children was "ungeheuer selten."

Dr. John Abercrombie, of Great Ormond street Children's Hospital, in London, believed it not frequent, though it did occur and was due to patent prepared baby foods. In a subsequent conversation with the house physician, in answer to the question—to what is scorbutus among children commonly attributed in this hospital—he promptly gave the name of a proprietary food which is advertised upon and within every omnibus in London, at every railway station in all England, and whose name glares at one from the back cover of every sixpenny novel. This point will be of interest to our worthy and honored

president. Well do we remember his able plea before the Academy of Medicine in New York, that the tender life of the children of this country might be emancipated from the thrall of enterprising pap-peddlers. It is a significant fact that the country which furnishes most of the literature of scorbutus in children is the same which is posted from end to end with advertisements of proprietary foods.

As to the frequency of scurvy in America, Dr. Roger S. Tracy, Register of Vital Statistics of New York Board of Health, has very kindly copied off for the writer all deaths reported under scurvy from 1870 to 1890. In this twenty years, there have been 100 deaths from scurvy, reported to the office, of which sixteen have been in patients of five years or under. Of the sixteen fatal cases in children, twelve have been in infants (under two years of age). So it comes about, that of the class of cases which we are here and now considering, sixteen have been reported among the deaths of the last two decades in the City of New York. Some have doubtless gone astray in the records, because scurvy has been little considered and less thought of among the derangements of childhood and infancy; many of them, no doubt, have found their way to the list of purpura and acute rickets, some to hæmorrhagic exanthemata.

Surgeon J. S. Billings, librarian of the Surgeon-General's office, has kindly furnished seven references to cases of scurvy in children and infants. The first five cases are from England, the sixth from France and the seventh from America. This case on examination is not considered scurvy by the writer.

It would seem that England, and that means London in this case, is the source of most of the cases, and of the literature, of scurvy in children. To W. B. Cheadle and Thomas Barlow of Great Ormond street Hospital, is due the credit (the former) of "having first shown on clinical grounds the true affinities of this form of infantile cachexia," and (the latter) the anatomical nature of the diseases determined by post-mortem examination. Scurvy

is not in the index of Meigs & Pepper's Diseases of Children (Edition, 1883) ; nor does it appear in the last edition of J. Lewis Smith's book.

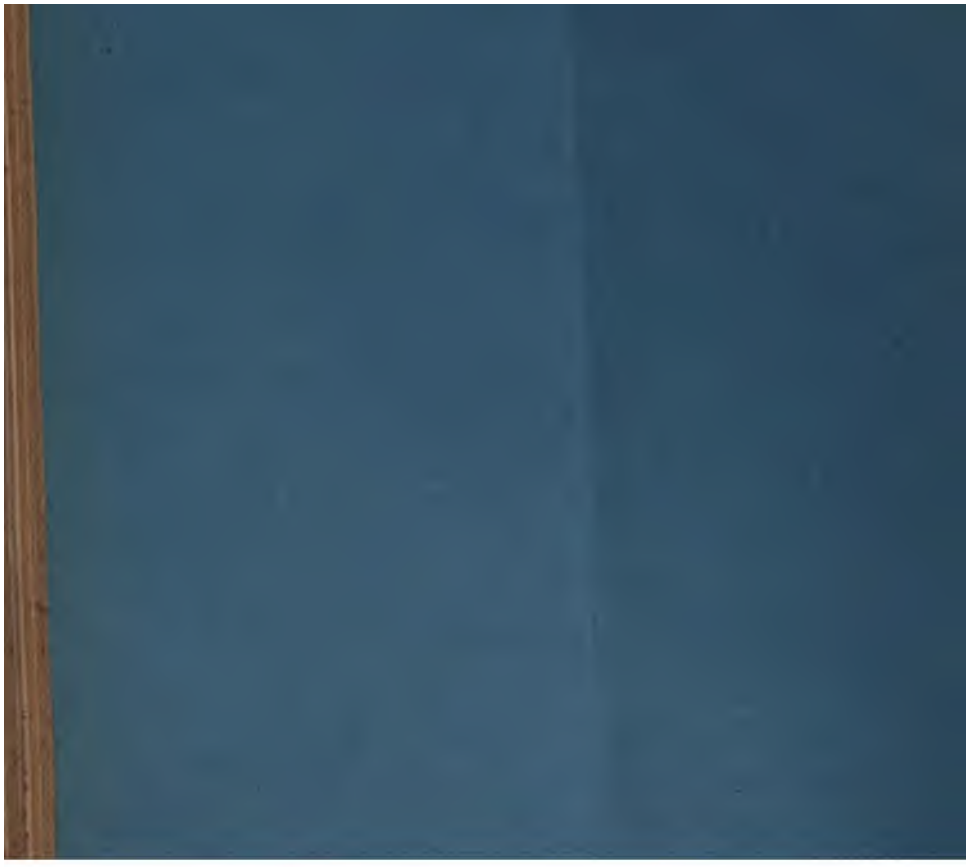
The writer has held correspondence with twenty-five persons, members of this Pediatric section, and others, in New York and different large cities ; the purpose has been to unearth any cases of infantile scurvy unreported and to learn the opinion of the gentlemen as to its frequency of occurrence. The answers have been prompt, to the point, of great service to the writer, and he here desires to express his obligations. All of these correspondents have vast opportunities for seeing cases in dispensaries, hospitals and asylums, as well as private practice. The majority of the answers contain such words (and with relief and satisfaction we record it) as "rare in children." "I know of very few cases of scorbutus" ; have thus far not seen any case of scurvy in children." "Have never recognized a case . . . nor do I know of any recognized case." "Have never seen a case in children in this city nor at the Juvenile Asylum where I pass in review 1,000 annually, and many are from the 'slums.'" "I think the cases extremely rare." "Cases of scurvy in children I have not seen." "During a service of two years, in which time 1,600 children have been seen, I have not met a single case of scurvy."

The purpose of this paper has been so modest that it may fairly be assumed that that purpose has been attained. Cases have been collected—eleven of them—and with pains to make the number represent the approximate frequency of occurrence. We must now await the report of the investigator for the intimate essential cause of the various lesions of scurvy. It is not a question of opinion; it is a question of facts. The whole line of sturdy soldiery must await the arrival of the aid who brings a message from him who holds his council amid culture ovens and flasks, dry plates and oil immersions.

So many apparent causative elements enter into the hæmorrhagic diseases and blood-dyscrasia, from drugs to bad food, that it seems idle to recount them ; they rest

just where they did decades ago. The germ investigations have not rendered a final answer, though they seem soon to explain some of the group.

Finally, to dwell on such a topic as the present, to bring together rather more cases of scurvy than one expects are to be found, to, in a certain way, apply for the admission of a new disease into American literature, is somewhat similar to the player's rôle of villain ; he may set forth upon the boards a most excellent villain, but after all, it is a gruesome business.



Aus der „Medicinischen Monatsschrift", Juni, 1889.

Lungenemphysem und Bronchialasthma im Kindesalter.

Von

Dr. Richard Stein,

Arzt am Deutschen Dispensary und am Hebrew Sheltering Orphan Asylum in New York.

Obwohl unsere Kenntnisse über die Entstehungsweise des Lungenemphysems der Erwachsenen noch sehr dürftig sind, so gehört das klinische Bild desselben zu den uns am besten bekannten Abschnitten der Pathologie. Der fassförmige Thorax, die starre inspiratorische Stellung desselben, der Tiefstand des Zwerchfells, die Ueberlagerung des Herzens durch die Lungenränder, das verlängerte Exspirium, das hypertrophische rechte Herz; die chronische Bronchitis des Emphysematikers, seine Kurzathmigkeit, die intercurrenten asthmatischen Anfälle — das sind alles Dinge die Jeder kennt. Und doch ist das Emphysem der Erwachsenen nicht so häufig als man gewöhnlich annimmt. *Virchow* fand in den Charitélisten in Berlin für die Jahre 1876—1885 im Ganzen nur 0,3% aller aufgenommenen Fälle bei denen die klinische Diagnose Emphysem angegeben war. Aus den Sectionslisten desselben Institutes ergaben sich 0,6—0,7% aller zur Section gelangten Fälle als solche, bei denen das Emphysem die Hauptkrankheit war.—Es braucht nur beiläufig erwähnt zu werden, dass hier unter Emphysem, Ueberausdehnung und Elasticitätsverlust des Gewebes zu verstehen sind, welche mit *Lungenschwund* einhergehen. Meist vergehen viele Jahre zwischen der ersten Entstehung des Processes und dem Tode.

Virchow ist geneigt eine Erkrankung im Lungengewebe selbst anzunehmen, die schon frühzeitig angelegt wird und sich dann im Laufe eines langen Zeitraumes, unter der Begünstigung mechanischer Momente allmälig entwickelt.

Das Lungenemphysem im Kindesalter und dessen verwandte Zustände haben, im Vergleich zu denen der Erwachsenen nur wenig Aufmerksamkeit von Seiten der Aerzte erfahren, trotzdem die Krankheiten die zum Emphysem führen sehr häufig sind, und trotzdem der ganze Process sich gewissermaassen unter den Augen des Arztes entwickelt. Das sonst unschätzbare Werk von *Henoch* (Vorlesungen über Kinderkrankheiten) — das allerdings nur eigene Beobachtungen enthält — schweigt ganz über diesen Gegenstand; andere Lehrbücher

erwähnen ihn nur mit einigen Worten. Auch begegnet man in der Literatur den grellsten Widersprüchen über das Lungenemphysem der Kinder; zum Theil liegt das an der Verwirrung in der Terminologie. Der Terminus „Emphysema pulmonum" ist ohne weiteres in die Pediatrik übertragen worden zur Bezeichnung gewisser acuter und subacuter Aufblähungszustände der Lunge, welche mit dem eigentlichen substantiellen Emphysem nur das gemein haben, dass sie zu einer Erweiterung der Lungengrenzen führen, mit dem Rückgang der ursprünglichen Krankheit (Pertussis, Pneumonie etc.) meist wieder ganz verschwinden.

Einige Autoren, namentlich englische und amerikanische, verstehen unter Emphysem im Kindesalter die schlechtweg als „Zellgewebsemphysem" bekannte, meist aus traumatischer Ursache entstandene Infiltration von Luft in's Unterhautfettgewebe.[1]) Klinische Angaben über Emphysem der Kinder bei den älteren Autoren fehlen vollends. Die Arbeiten von *Hewitt*, *Hecker* und *Hervieux* geben wohl genaue anatomische Beschreibungen der an Kindesleichen des frühesten und frühen Alters vorgefundenen emphysematösen Veränderungen, doch fehlen dabei die klinischen Befunde. *Hewitt* sagt: In der That ist in der ersten Lebenszeit keine krankhafte Veränderung der Lunge häufiger als das Emphysem. Nach ihm sind besonders die oberen Lappen beider Lungen zu Emphysem geneigt, und namentlich deren vordere Abschnitte. Auch hat er niemals Apneumatose (Atelectase) gesehen, ohne gleichzeitiges Emphysem. Im Uebrigen erstrecken sich seine Erfahrungen nur auf Kinder bis zu drei Jahren.

Hecker beschreibt einen Fall von Lungenemphysem eines neugeborenen Kindes, welches in utero entstanden ist. Der Fall ist höchst bemerkenswerth und kann einer scharfen Kritik Stand halten; er ist von gerichtsärztlichem Standpunkte aus von grossem Interesse.

Von den siebenundzwanzig hierhergehörigen Fällen die *Hervieux* gesammelt hat, stammen neunzehn aus dem Alter vor dem zwanzigsten Lebenstage. Das allgemein verbreitete Emphysem hat er niemals beobachtet.

Rilliet und *Barthez* sprechen sich schon positiv über das Vorkommen von verbreitetem vesiculären Emphysem bei Kindern aus und geben die klinischen Zeichen desselben mit gewohnter Schärfe der Beobachtung wieder. Sie weisen namentlich auf den Zusammenhang von rhachitischen Deformationen des Brustkorbs mit Emphysem hin, sowie auf dessen Vorkommen bei acuten Krankheiten der Respirationswege, wobei sie hauptsächlich den Larynxcroup und die verschiedenen Arten der Pneumonie im Sinne haben. Sie geben ferner an, dass sie bei Kindern von fünf bis sechs Jahren Fälle von stationärem substantivem Emphysem beobachtet haben, mit all den characteristischen Begleitsymptomen wie sie bei Erwachsenen vorkommen, vom pfeifenden ver-

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[1]) Siehe The Index of the Library of the Surgeon-General of the United States Army.

längerten Exspirium bis zur starren inspiratorischen Stellung des Brustkorbes. Auch litten diese Kinder — es waren deren fünf — an asthmatischen Anfällen.

Steffen, dem ein grosses klinisches und anatomisches Material zu Gebote steht, gibt die erste ausführliche Beschreibung des Emphysems der Kinder. Er richtete seine Aufmerksamkeit ganz speciell auf diese Krankheit. Nach ihm kommt das Emphysem ausserordentlich häufig im kindlichen Alter vor; bei etwa drei Vierteln der von ihm gemachten Sectionen kindlicher Leichen fand er Emphysem in grösserer oder geringerer Ausdehnung. Auch kommt nach ihm das Emphysem in den ersten Lebensjahren überwiegend häufiger vor als später. Diese Angaben sind höchst bemerkenswerth und fordern zu weiterer Beobachtung auf. Entgegen der Angabe von *Rilliet* und *Barthez* findet er Emphysem als constante Begleiterscheinung beim Keuchhusten; dafür hat er klinische sowie auch anatomische Belege. Ferner betont *Steffen* das mechanische Moment in der Entstehung des Emphysems; es wird hauptsächlich verursacht durch stossweise ausgeführte, aussetzende, krampfhafte Athemzüge (Husten u. dergl. m.). Dieser Factor ist es auch, welcher dem agonalen Emphysem zu Grunde liegt.

Schliesslich ist noch die Arbeit von *Fürst* im *Gerhardt*'schen Sammelwerk zu erwähnen, welche den jetzigen Standpunkt unserer Kenntnisse dieser Zustände darlegt.

Es wird wohl Keiner einen Augenblick im Zweifel darüber sein, dass die obengenannten Autoren, wenn sie das Wort Emphysem gebrauchen, einerseits das *vicariirende* Emphysem darunter verstehen; das sind Aufblähungszustände der relativ intacten Lungenparthien, welche sich bei acuten oder subacuten Entzündungsprocessen der Lungen entwickeln und parallel mit denselben verlaufen; und andererseits meinen sie damit das zuerst von *Traube* und später von *Cohnheim* sobenannte *Volumen pulmonum auctum;* das ist eine, gewöhnlich weit verbreitete Aufblähung der Lungenalveolen, deren Entstehung nur auf rein mechanische Momente zurückzuführen ist; dieselbe mag vielleicht Circulationsstörungen hervorbringen; diese sind aber nur vorübergehend, wie das Volumen pulmonum auctum selbst, und schliesslich — und das ist die Hauptsache — geht dasselbe, soweit uns bekannt, nicht wie das substantive Emphysem mit Lungenschwund einher. Es fällt dieser Begriff des Volumen pulmonum auctum so ziemlich mit dem der *Lungenblähung* von *Biermer* zusammen.

Leider ist über den Zusammenhang zwischen dem rein mechanischen Emphysem, das sich ja so häufig im Kindesalter findet, und dem substantiven (vesiculären) Emphysem des späteren Kindesalter und der Erwachsenen so gut wie nichts bekannt. Und doch muss ein Zusammenhang bestehen. *Virchow* betonte erst vor Kurzem, dass die Entstehungsgeschichte des Emphysems sehr häufig in das frühe Kindesalter zurückzuführen ist; er kennt gewisse albinistische Zustände der Lunge die sich mit Emphysem zusammenfinden, — ein Wahrscheinlichkeitsbeweis dafür, dass das Emphysem noch vor dem fünften Lebens-

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jahr zurückdatirt, wo bekanntlich die Lunge frei von Kohlenstaub ist.

Wie das Emphysem klinisch durch die Verbreiterung der Lungengrenzen erkannt wird (*Skoda*), ebenso das Volumen pulmonum auctum. Als Paradigma des letzteren möchten wir die Ausdehnung der Lungen beim Keuchhusten hinstellen.

Die Lungengrenzen der Kinder verhalten sich nach den eingehenden Studien *Weil's* und *C. Schmidt's* so, dass dieselben überall einen halben und selbst einen ganzen Intercostalraum höher stehen als die der Erwachsenen; das ist der *kindliche Typus* der unteren Lungengrenzen. Je jünger das Kind desto ausgeprägter ist derselbe. Ferner ist die relative Herzdämpfung bei Kindern eine verhältnissmässig grössere und mehr markirte, als im höheren Alter. Sie entspricht beinahe der Grösse des kindlichen Herzens, indem sie sich nach links häufig über die linke Mammillarlinie erstreckt, und nach rechts im concaven Bogen über das Sternum verläuft *(Weil)*. Dazu kommt noch, was schon lange bekannt ist, dass beim Keuchhusten das Volumen pulmonum auctum wesentlich ein Randemphysem darstellt, und schliesslich dass sich meist zuerst die Lingula der linken Lunge — überhaupt ein Locus minoris resistentiae in den Erkrankungen dieses Organs — im Zustande der Aufblähung befindet. Somit schlagen sich die Lungenränder über das Herz, und häufig bedecken sie dasselbe vollständig. Entgegen der Annahme von *Rilliet* und *Barthez* und übereinstimmend mit den Angaben aller Anderen, können wir das Vorkommen der Lungenblähung bei der grossen Mehrzahl der Fälle von Tussis convulsiva, allerdings nur von klinischer Seite bestätigen. Nahezu regelmässig ist dasselbe im Stadium convulsivum und darüber hinaus zu constatiren. Häufig besteht diese Lungenblähung wochenlang nach der Dauer der Pertussis selbst; der Percussionsschall am Thorax ist lauter und voller als sonst, die Lungengrenzen sind nach unten verschoben, und die Herzleere ist schwer oder gar nicht zu bestimmen. Allmälig verschwindet, wenigstens den klinischen Zeichen nach, das Volumen pulmonum auctum, und es ist anzunehmen, dass die Lunge in den allermeisten Fällen, was ihren Umfang und ihre Elasticität anbetrifft, auf den Status quo ante zurückkehrt. Die Kinder sind dabei gewöhnlich kurzathmig, und abgesehen von der während der Acme der Tussis häufig complicirenden Pneumonien, gesellen sich leicht Bronchitis und Bronchopneumonie nach Ablauf des eigentlichen Keuchhustens hinzu. Nichts liegt daher näher als die Annahme, dass sich die Respirationsschleimhaut als auch das Lungengewebe selbst in einem Desintegrationszustand befindet.[1]) Meist kehrt Alles wieder zur Norm zurück. Inwieweit dieser Zustand geneigt ist permanent zu werden und die Grundlage zum späteren Emphysem zu liefern, darüber wissen wir nichts Positives.

Die grössere Elasticität des Lungengewebes im Kindesalter bringt es mit sich, dass auch noch andere mehr acut verlaufende Processe als es der Keuchhusten ist die Lungenblähung zu Stande kommen lässt.

[1]) Das Auftreten von Lungenphthise nach Pertussis ist nichts Ungewöhnliches.

Hierher gehört das Asthma bronchiale, welches für das Kindesalter zuerst von *Politzer* statuirt worden ist, dem aber seitdem (1870) so gut wie keine Aufmerksamkeit von Seiten der Kinderärzte geschenkt wurde.

Ueber die Natur des Asthma bronchiale überhaupt, sind die Acten noch nicht geschlossen. Seitdem *Romberg* zuerst das Bild des Asthma nervosum zeichnete, indem er dasselbe als einen reinen Reflexvorgang auffasste, schwankte dessen Stellung in der Pathologie fortwährend. Leider muss zugestanden werden, dass die Bestrebungen der neueren Zeit [1]) dem Asthma bronchiale eine genaue anatomische resp. physiologische Basis unterzulegen, als unzureichend anzusehen sind. Die histologischen Untersuchungen der Sputa beim Bronchialasthma haben manchen interessanten Befund aufgedeckt, wie z. B. die *Charcot-Leyden*'schen Crystalle und die *Curschmann*'schen Fibrinpfröpfe (die übrigens schon früher bekannt waren), das Wesen des Asthma bronchiale aber lässt sich wie vorher nur vermuthen. Der (hypothetische) Zwerchfellkrampf *(Wintrich-Bamberger)* ist längst verlassen worden. Nur *Riegel* tritt neuerdings wieder für denselben ein, nachdem er ihn lange bekämpft hatte. Das Hinzuziehen der *Leyden*'schen Asthmacrystalle zur Erklärung des Zustandekommens der asthmatischen Anfälle ist von *Leyden* selbst und allen Anderen als nichtig anerkannt worden; die Crystalle finden sich auch sonst noch bei manchen Bronchitiden, bei Pneumonien, beim Lungenbrand. Ferner treten dieselben auffälligerweise in grösster Anzahl erst *nach* dem Anfall im Sputum auf. Nimmt man wie *Ungar* es thut eine "capilläre fibrinöse Bronchitis" oder wie *Curschmann* eine "Bronchiolitis exsudativa" als Grundlage des Bronchialasthma an, so zeigt das eben nur, dass man dasselbe nicht als Krankheit sui generis betrachten kann, sondern nur als ein intercurrirendes Symptom gewisser pathologischer Zustände der Bronchialschleimhaut. Denn die Bronchiolitis an sich genügt doch nicht um den asthmatischen *Anfall* — und darauf kommt es doch an — zu erklären. Ueber diesen Punkt wurde man sich auch so ziemlich einig. Von diesem Gesichtspunkte aus hat aber die alte Theorie des Catarrhus acutissimus *(Fraentzel-Waldenburg)* ebensoviel Berechtigung, als irgend eine andere. Für das Kindesalter erscheint dieselbe uns am meisten zutreffend. [2]) Zur Erklärung des Anfalls selbst müssen wir wieder auf den Brochialkrampf *Biermer's* zurückgreifen. Eine wesentliche Stütze erhält letztere Ansicht noch durch die Experimente von *MacGillavry* [3]) und von *Riegel-Edinger.* [4])

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[1]) Siehe namentlich die Verhandlungen des Congresses für Innere Medicin, Wiesbaden, 1882 und 1884.

[2]) *Trousseau* hat schon längst auf die Bedeutung des Catarrhs als aetiologisches Moment beim Asthma aufmerksam gemacht.

[3]) *MacGillavry*, Nederl. Tjidschrift voor Geneeskunde 1876 und *Donders*, Discussion über Bronchialasthma-Verhandlungen d. Congresses f. Innere Medic., Wiesbaden 1885.

[4]) *Riegel-Edinger*, Zeitschrift f. klinische Medic., Bd. 5, p. 413.

Das Bronchialasthma stellt also ein Symptom dar das plötzlich auftritt um meist ebensoschnell wieder zu verschwinden. Die zugrundeliegenden Krankheiten können dabei die verschiedensten sein: Emphysem, Bronchitis, Bronchiolitis, gewisse fluxionäre Zustände der Nasen- und Rachenschleimhaut, und schliesslich gewisse grobanatomische Veränderungen und Neubildungen der ersten Respirationswege, als da sind, Hypertrophien der Nasenmuscheln, Anomalien des Septum narium, Hypertrophie der Tonsillen, Polypen u. dgl. m. Ueber die primäre Fluxion (*Weber-Stoerck*) kann weiter kein Zweifel sein, nachdem *Stoerck* direct mit dem Kehlkopfspiegel zum Beginne des Anfalls eine stark auffallende Hyperämie des Kehlkopfs und der Trachea beobachtete. Die catarrhalischen Zustände beim Heufieber mit den consecutiven asthmatischen Anfällen sind bekannt. — Vor einiger Zeit behandelten wir eine Dame, die periodisch (nicht nur im Spätsommer) an typischen Asthmaanfällen litt, die jedesmal mit einem Schnupfen anfingen, des Nachts exacerbirten, und mit abundanter Schleimabsonderung aus beiden Nasenhöhlen einhergingen. Nach Aussage der Patientin möchte sie das Asthma gern ertragen, wenn ihr nur nicht die heftige Schleimabsonderung aus der Nase grosse Umstände bereiten würde. Die Untersuchung der Nase ergab stark hypertrophische untere Nasenmuscheln, welche in mehreren Sitzungen mit dem Galvanocauter zerstört wurden. Es wurden die Anfälle leichter, dieselben verschwanden aber nie ganz.

Es spielt somit das fluxionäre Element, resp. der Catarrhus acutissimus die Hauptrolle in der Genese des Anfalls. Das eigentliche Wesen desselben soll in einem Reflexvorgang bestehen, hervorgerufen durch die Erregung der peripheren Endigungen des Trigeminus, resp. Vagus. In den Bahnen des letzteren kommt sodann der Bronchialkrampf zu Stande. Inwieweit abnorme Erregung des Zwerchfells mitwirkt bleibt unentschieden.

Das eigentliche Analogon des Asthma bronchiale der Erwachsenen scheint im Kindesalter weniger das Asthma selbst zu sein, als vielmehr eine Erkrankung die ungemein häufig bei Kindern auftritt und uns häufig unsere Nachtruhe stört; das ist der Pseudocroup. Derselbe beruht auch primär auf einem acuten Catarrh des Larynx und wird schlechtweg als solcher aufgefasst. Catarrhalische Symptome gehen zumeist voraus, der Croupanfall schliesst sich denselben aber erst an. Das plötzliche oft unerwartete Auftreten desselben, meist inmitten tiefen Schlafs, das Wiederkehren des Anfalls, die heftigen dyspnoëischen Erscheinungen bei geringer Fieberbewegung oder gar Fieberlosigkeit; das ebenso schnelle Verschwinden des Anfalls; der Ausgang in einen Bronchialcatarrh mit hohem pfeifenden Exspirium, sind alles Erscheinungen die ceteris paribus mit dem Bronchialasthma grosse Aehnlichkeit haben. Der Catarrh allein genügt aber nicht zur Erklärung aller dieser Symptome. Es ist mehr als wahrscheinlich, dass ebenso wie beim Bronchialasthma gewisse spastische Momente hinzutreten um den Croupanfall auszulösen.

Nur vorübergehend wollen wir hier bemerken, dass noch ein weiterer Factor hinzukommt der in der Discussion über Asthma völlig übersehen wurde, der aber, so vage auch dessen Begriff ist, und so wenig positive Unterlage derselbe zur Zeit besitzt, ein wichtiges Agens in der Aetiologie der Lungenkrankheiten darstellt: die *Disposition*. Dieselbe ist entweder hereditär: es gibt ganze asthmatische und emphysematöse Familien,[1]) oder sie ist erworben. In letzterer Beziehung sind gewisse Krankheiten zu erwähnen, wie Masern, Keuchhusten und die Rhachitis.

Kinder sind überhaupt sehr zu Catarrhen disponirt und wir sehen asthmatische Zustände sich nicht so selten zu acuten Catarrhen der Luftwege hinzugesellen. Hierher gehören die von *Rilliet* und *Barthez* beschriebenen Affectionen der Kinder, welche „durch ihre kurze Dauer, ihre häufige Widerkehr, die Intensität der Dyspnoë, zugleich aber auch durch den geringen Fiebergrad sich den asthmatischen Anfällen Erwachsenen nähern." *Henoch* hat eine grössere Anzahl solcher Fälle schon bei kleinen Kindern gesehen, häufiger aber in der zweiten Periode der Kindheit; die Kinder litten schon seit Jahren an diesen Anfällen. *Henoch* will diese Krankheit als „recidive Bronchitis" benannt wissen. Das Krankheitsbild ist folgendes: Ein Kind das an einem Schnupfen leidet bekommt plötzlich, mit oder ohne Erscheinungen eines Pseudocroup, hochgradige in- und exspiratorische Dyspnoë, mit beschleunigtem Athmen und äusserst frequentem Puls, bis zu den hochgradigsten Stickanfällen. Die Ergebnisse der Auscultation des Thorax sind verschieden bei verschiedenen Kindern, bald ist Schleimrasseln zu hören, bald nicht. Diese Anfälle können sich wiederholen. Von einer Bronchitis capillaris kann hier nicht die Rede sein; der Anfall ist gewöhnlich schon innerhalb einiger Stunden vorbei. Es ist nicht erwähnt ob sich Lungenblähung dabei ausbildet.

Wir können zwischen diesem Krankheitsbilde und dem von *Politzer* unter den Namen Bronchialasthma, Bronchienkrampf im Kindesalter, beschriebenen Beobachtungen keinen principiellen, sondern nur einen graduellen Unterschied finden. Bei Letzterem war auch zuerst Bronchitis vorhanden, mit auffallend asthmatischem Character; später traten dann die entzündlichen Erscheinungen mehr zurück, und es schlossen sich dann asthmatische Anfälle in mehr typischer Weise an. Zwischen den Anfällen bestand meistens noch Bronchialcatarrh. *Politzer* legt Nachdruck auf das hohe feine Pfeifen, welches allenthalben am Thorax im Anfall zu hören ist. Dasselbe soll sich namentlich auf die Exspiration beschränken und für Asthma bronchiale characteristisch sein. Wenn wir nicht irren, ist das dasselbe Exspirationsgeräusch, welches wir im Englischen unter "wheezing" verstehen. — Vor einiger Zeit behandelten wir ein zwölf Monate altes Kind, welches im dritten Monate schon Anfälle von Asthma bekam, welche sich viermal nacheinander alle drei Wochen wiederholten. Zur Zeit als es in unsere Beobachtung kam, hatte es eben wieder einen Anfall hinter sich. Die

[1]) Siehe Ehebald, D. med. Wochenschr., 1881, VII, p. 137.

Auscultation des Thorax ergab hohes feines Pfeifen nebst gross- und kleinblasigen Rasselgeräuschen; die Percussion eine deutliche Lungenblähung; die Lungengrenzen waren nach allen Richtungen hin verbreitert; der Herzstoss fehlte, und absolute sowie relative Herzdämpfung konnten nicht bestimmt werden. Die Inspiration vollzog sich mit einer deutlichen Einziehung, entsprechend der Insertion des Zwerchfells. Diese als „peripneumonische Furche" bekannte Einziehung des unteren Theils des Brustkorbs kommt auch normaliter zur Beobachtung, doch fehlt sie nie bei der Dyspnoë im Kindesalter. Wir kommen auf dieses verschiedentlich als Flankenschlagen, peripneumonische Furche, Harrison'sche Fursche benannte Phänomen, sowie auf dessen Folgezustände noch weiter unten zurück. — Das Kind, um welches es sich hier handelt, war rhachitisch und machte noch keine Gehversuche. Der Thorax zeigte starke Difformität: es war die so häufig vorkommende Verbiegung im unteren Drittel desselben vorhanden. Nebenbei bestand aber eine starke Einziehung. Verschiedentliche Messungen ergaben: Der Brustumfang in der Höhe der Brustwarzen betrug 47 cm.; unterhalb derselben nur 45½ cm. Misst man in derselben Höhe über den Thorax von hinterer Axillarlinie der einen Seite zur hinteren Avillarlinie der anderen Seite so beträgt das Maass 28 cm. Weiter unten am Thorax in der grössten Tiefe der Einziehung beträgt das Maass zwischen denselben Punkten nur 25 cm. Die Differenz bezieht sich also im Wesentlichen auf den vorderen Abschnitt des Thoraxumfangs. Im Uebrigen ist der Uebergang von der hinteren zur vorderen Hälfte des Brustkastens ein ziemlich schroffer; die Rippen sind an dieser Stelle winklig nach vorne abgeknickt.

Die Configuration des Thorax im kindlichen Alter ist eine mannichfaltige. Sehr häufig, namentlich bei rhachitischen Kindern findet man Verbiegungen des Manubrium und Corpus sterni, Schrägstellung und Abknickung des Processus ensiformis, ferner Eindrücke der Rippen vorzugsweise in der Gegend der unteren Thoraxapertur, abgesehen von den ganz groben Veränderungen, die als kielförmiger Thorax, Pectus carinatum, Hühnerbrust bekannt sind; ferner Heteromorphien verursacht durch Scoliosen, Kyphosen u. dgl. m. Auch hängt die Form des Thorax vor allen Dingen von der stärkeren oder schwächeren Entwickelung der Muskel und des Fettpolsters ab. Die Kleidung, der Füllungszustand der Brust- und Baucheingeweide haben ferner einen directen Einfluss auf die Form des Thoraxskelettes.

Die Einbiegungen an den unteren wahren Rippen sind ein häufiges Vorkommniss; dieselben erstrecken sich manchmal bis zur Axillarlinie. Von dieser Einbiegung steht dann der Rippenbogen ziemlich steil nach vorn, um sich compensatorisch den, im Kindesalter relativ stark angefüllten, Baucheingeweiden anzupassen. Die Entstehung dieser Eindrücke ist noch nicht genügend aufgeklärt.

Beim Emphysem, vorzüglich aber während des asthmatischen Anfalls, sind Expiration und Inspiration insufficient. Die Lunge ist ad extremum aufgebläht; auch kann sich theilweise Atelectase aus-

bilden. Das Zwerchfell wird tief herabgedrängt, und reagirt hierauf mit unregelmässigen krampfhaften Contractionen. Bei der Inspiration werden alle accessorischen Athmungsmuskel am Halse und am Thorax in heftigste Action versetzt. Die Rippenansätze des Zwerchfells folgen letzterem bei jeder Contraction und es bildet sich daher bei der Inspiration die peripneumonische Furche aus. Im Volksmund wird das als Flankenschlagen bezeichnet.

Leidet ein Kind an heftigen Asthmaanfällen mit sich daran anschliessender Lungenblähung, so wird die Furche immer mehr markirt. Hat man es mit weichen rhachitischen Knorpeln und Knochen zu thun (und das ist ja meist der Fall) so folgen dieselben dem Zug des Zwerchfells und die peripneumonische Furche wird permanent.

Dieselbe ist nicht wie *Steffen* behauptet geradezu pathognomonisch für Emphysem. Dieselbe kommt auch vor bei Kindern die von Geburt an chronischen Bronchitiden leiden; ferner wurde sie auch von *Gerhardt* für die erworbene Lungenatelectase beansprucht. Im letzteren Fall ist dieselbe anzusehen, als das Bestreben des Thorax sich den nichtausdehnungsfähigen (atelectatischen) Partien der Lungen anzupassen.

Den geringsten Widerstand bieten natürlich die weichen Knorpel. Daher ist die Einziehung entsprechend der Mitte der unteren Thoraxapertur am meisten ausgeprägt. Der Processus ensiformis kann zu gleicher Zeit stumpfwinklig abgeknickt sein. Die von uns beobachteten Kinder, welche diese Verhältnisse darboten waren rhachitisch. Ob die permanente peripneumonische Furche constant bei rhachitischen Kindern vorkommt, ferner wie sich die Verhältnisse bei nicht rhachitischen emphysematoesen Kindern gestalten, müssen weitere Beobachtungen lehren. Fälle von fassförmigem Thorax bei älteren Kindern sind von *Rilliet* und *Barthez*, *Waldenburg*, *Ehebald* beschrieben. Ueber fassförmigen Thorax bei ganz jungen Kindern existiren keine Beobachtungen. Einen solchen Fall hat Herr Dr. Seibert beobachtet und ihn in der wissenschaftlichen Zusammenkunft deutscher Aerzte in New York vorgestellt. Wir lassen die Krankengeschichte des Falles, welche uns Herr Dr. Seibert in freundlicher Weise zur Verfügung stellte, hier folgen: „Es handelt sich um einen Fall von allgemeinem stationären Vesiculäremphysem bei einem Kinde von 14 Monaten. Laut Angabe der Mutter bekam das Kind im Alter von 14 Tagen Rasseln auf der Brust und darauf schweres Athmen. Diese Erscheinungen hielten sich von März 1883 bis zum Sommer desselben Jahres, als entschiedene Besserung eintrat. Im Herbst folgte wieder Verschlimmerung, welche bei feuchter Witterung und nach jedem Gang in die freie Luft besonders stark wurde. Anfangs December soll das Kind eine Lungenentzündung durchgemacht haben, die mehrere Wochen anhielt. Nach Ablauf dieser Complication bestand das Rasseln und mehr oder minder grosse Athemnoth weiter fort. Sonst hatte das Kind eine gute Verdauung, und wurde kräftig und stark. Auffallend war nur der ‚dicke Leib' des Kindes. Am 20. Februar 1884 brachte die Mutter das Kind in die Kinderabtheilung des Deutschen Dispensary. Der Status war folgender:

Ein kräftig genährter, gut entwickelter, zwölf Monate alter Knabe; blühende Gesichtsfarbe. Temperatur normal. Puls und Respiration beschleunigt. Dabei erscheint das Kind nicht schwerkrank, sondern benimmt sich fidel und munter. Beim Auskleiden fällt sofort der grosse Umfang des Thorax und des Leibes in's Auge. Die Form des Thorax ist die als Fassform bekannte. Die Zwischenrippenräume sind gross. An der beschleunigten Athmung betheiligten sich alle accessorischen Athmungsmuskel. Die Percussion ergibt hellen Lungenschall bis zur zehnten Rippe herab, auf beiden Seiten des Thorax, bei vollständigem Fehlen der Herzdämpfung. Die Leber und alle übrigen Baucheingeweide sind durch das ausserordentlich tiefstehende Zwerchfell derartig nach unten gedrückt, dass der grosse Bauch des Kindes dadurch sofort seine Erklärung findet. Die Auscultation ergibt überall Vesiculärathmen mit theils trockenen, theils feuchten gross- und kleinblasigen Rasselgeräuschen, und ausserdem hohes feines Pfeifen bei der Exspiration. Die Herztöne sind kaum hörbar wegen den Geräuschen in der das Herz bedeckenden Lungenpartie.

In diesem Fall also setzte zwei Wochen nach der Geburt des Kindes ein Catarrh der feinen Bronchien der ganzen Lunge ein, und blieb ein volles Jahr bestehen. — Jetzt ist das Kind 14 Monate alt. Sie sehen einen immerhin noch sehr weiten Thorax, bedeutendes Tiefstehen der Lunge, aber absolut keinen Catarrh irgend welcher Art. Auch in diesem Fall hat die Wegnahme der Ursache des Emphysems verbunden mit dem Wachsthum des Kindes bei guter Ernährung und Pflege eine theilweise Rückbildung geschaffen. —

Letzthin kam uns folgender Fall zur Beobachtung: Ein fünfjähriger Knabe, der sonst immer gesund war und aus gesunder Familie stammt, bekam im Sommer vorigen Jahres zusammen mit seinen anderen Geschwistern den Keuchhusten; derselbe war recht heftig. Im letzten Winter acquirirte er hämorrhagische Masern, die mit hoher Temperatur und bedeutendem Bronchialcatarrh verliefen. Hierauf relatives Wohlbefinden. Einige Wochen darauf, wieder heftige Bronchitis mit asthmaartigen Anfällen, die namentlich des Nachts auftraten. Dabei entwickelte sich Lungenblähung. Bei der Auscultation hörte man rauhes Athmen, und alle Zeichen eines Bronchialcatarrhs. — Ausgeprägtes typisches Bronchialasthma war nie dabei vorhanden, sondern nur Anfälle von Kurzathmigkeit. Die Zeichen des Catarrhs und der Lungenblähung verschwanden allmählig ganz.

Die Entwickelung der Lungenblähung nach den heftigen hämorrhagischen Masern ist sehr interessant. —

Folgender Fall von Lungenemphysem, der jetzt noch, nach drei Jahren besteht, und den wir unter unseren Augen entstehen sahen, ist von speciellem Interesse, da er sich zweifellos aus wiederholten heftigen Attacken von Bronchialasthma entwickelte. Der Fall ist seit drei Jahren in unserer Beobachtung. Es mag desswegen eine etwas ausführlichere Beschreibung erlaubt sein:

A. S. ein siebeneinhalb Jahre altes Mädchen, das dritte Kind

gesunder Eltern; die zwei älteren Geschwister als auch das jüngste Schwesterchen sind ganz gesund. In der Familie lässt sich keine Tendenz zu Emphysem oder Asthma nachweisen. Das Kind wurde von der Mutter selbst genährt. Kein Sommercomplaint. Mit vierzehn Monaten fing sie zu gehen an. Mit fünfzehn Monaten überstand sie eine leichte Lungenentzündung. Vom Säuglingsalter an war sie zu Bronchialcatarrhen geneigt, namentlich bei windigem Wetter. Sonst war aber ihr Gesundheitszustand ein recht guter. In ihrem fünften Jahr (Mai 1886) hatte sie wiederum eine leichte Erkältung, Schnupfen und Bronchialcatarrh mit leichten Fieberbewegungen. Am zweiten Tag dieser Erkrankung nahm das Rasseln auf der Brust zu, das Kind schlief in der darauffolgenden Nacht sehr unruhig. Gegen Morgen stellte sich stark beschleunigtes Athmen ein, mit weit hörbaren Rasselgeräuschen; binnen einigen Stunden entwickelte sich dann die hochgradigste Dyspnoë. Die kleine Patientin lag da, nach Athem ringend, mit äusserst frequentem kleinen Puls, kalten Extremitäten, die Haut mit klebrigem Schweiss bedeckt, ausgesprochene Theilnahmslosigkeit. Man hörte schon beim Eintritt in's Zimmer, sehr laute, feuchte Rasselgeräusche, die bei der unmittelbaren Auscultation gleichmässig über den Thorax verbreitet zu sein schienen. *In diesem Anfall, sowie auch in den späteren war kein Fieber vorhanden.* Dieser erste Anfall war so heftig, dass man sogar auf den Exitus lethalis gefasst war. — Allmählig hob sich die Dyspnoë; das Kind hustete in kurzen Intervallen, und unter den Erscheinungen eines Bronchialcatarrhs endete diese Erkrankung mit Genesung innerhalb einiger Tage. — Der Anfall wiederholte sich unter etwas minder stürmischen Erscheinungen einen Monat später (19. Juni.) Das Kind erholte sich dann rasch. Im Spätjahr 1886 trat wieder ein Anfall auf, der sich dann alle zwei bis drei Monate wiederholte. Nach Aussage der Mutter stellen sich die Anfälle nach heftigeren Bewegungen ein, Springen u. s. w. Im Frühjahr 1887, wieder ein heftiger Anfall, ebenso im Juni. Hierauf ging das Kind nach Europa, und hielt sich zuerst im Schwarzwald auf, wo es an Bronchialcatarrhen litt. Während seines Aufenthalts in Europa wurden ihm die Rachentonsille entfernt. — Seit ihrer Rückkehr, hatte sie nur zwei leichte Anfälle. Seit letztem Sommer erfreut sie sich guter Gesundheit; seit einem Jahr etwa sind die Asthmaanfälle ganz ausgeblieben.

Status praesens: Ein seinem Alter entsprechend grosses Mädchen, mit gering entwickeltem Fettpolster. Leichte Kopf- und Kieferrhachitis. Zähne unregelmässig, zackig. Die Knochen der Ober- und Unterextremitäten im Schaft leicht gebogen. Epiphysenauftreibung leichten Grades.

Der Thorax selbst ist keineswegs fassförmig. Er ist eher länger als gewöhnlich. Die Fossae supra- und infraclaviculares deutlich markirt. Auffallend ist eine Einziehung die sich der ganzen Länge des Brustbeins entsprechend von der Fossa jugularis bis zum Schwertfortsatz herunterzieht und in der Gegend des Schwertfortsatzes selbst eine tiefe Grube bildet (permanente peripneumonische Furche). Der Schwertfortsatz im stumpfen Winkel abgeknickt. Der Brustumfang in dieser Gegend

ist etwa zweieinhalb Centimeter geringer als der Brustumfang in der Höhe der Brustwarzen. Die Lungengrenzen überschreiten die der Norm beträchtlich: In der Mammillarlinie an der 8ten Rippe; in der Axillarlinie an der oberen Grenze der 9ten Rippe; in der Scapularlinie im 10ten Intercostalraum. Die unteren Lungengrenzen sind frei, nicht adhärent. — Die Auscultation ergibt zur Zeit überall vesiculäres Athmen. — Der Herzstoss ist weder sichtbar, noch durch die Palpation vernehmbar. Die Herzleere selbst ist nur angedeutet; die absolute Herzdämpfung ergibt einen Dämpfungsstreifen entsprechend der Stelle des sechsten und siebenten Rippenknorpels. — Herztöne sind rein. Sonstige Organe normal.

Wir hatten Gelegenheit verschiedene asthmatischen Anfälle mit zu beobachten, und konnten uns davon überzeugen, dass wir es mit einem Fall von *typischem Bronchialasthma* zu thun hatten. Die Dyspnoë war in- und exspiratorisch, vornehmlich das letztere. Die Athemzüge waren höchst frequent, bis vierzig und darüber in der Minute. — Beiläufig bemerkt wird allgemein die Respirationsanzahl im asthmatischen Anfall der Erwachsenen als verringert oder wenigstens doch nicht vermehrt angegeben; diese Verminderung wird auf die verlängerte Exspiration bezogen. Das mag regelmässig so sein. Doch behandelten wir vor zwei Jahren einen alten Herrn mit sehr ausgedehntem Emphysem, der an häufig wiederkehrenden heftigen Asthmaanfällen litt. Zu Beginne der Anfälle war stets die Respirationsanzahl vermehrt; nachdem die Dyspnoë geringer wurde, das Herz sich einigermassen erholte, kehrte die Zahl der Athemzüge wieder zur Norm zurück. — Dieser Fall mag eine Ausnahme sein; wie dem aber auch sei, im Kindesalter scheint die Respirationsanzahl im asthmatischen Anfall vermehrt zu sein. Damit stimmen auch die Beobachtungen von *Politzer*.

Die physikalischen Zeichen im Anfall sind kurz folgende: Ungemein beschleunigtes Athmen; hochgradige Action aller accessorischen Inspirationsmuskel, Cyanose der Lippen, der Finger, der Fusszehen. Distension der Schlüsselbeingruben und Intercostalräume, scharf markirte Einziehung an der unteren Parthie des Thorax bei der Inspiration; brettharte Contraction der Bauchmusculatur bei der Exspiration (accessorische Action der Recti abdominis). Verfallene Gesichtszüge, Benommensein des Sensorium; rastloses Umherwerfen. Erhöhte Lage gibt wenig Erleichterung.—Der Puls jagend und unregelmässig.— Von Weitem schon hörbar, ein sägendes, rauhes, feuchtes Rasselgeräusch, das am Ende des Exspirium mit hohem, feinen Pfeifen vergesellschaftet ist. Sonstige Athemgeräusche sowie Herztöne etc. werden durch dieses Geräusch verdeckt. — Die Percussion ergibt hellen, lauten Schall; keinen Schachtelton. Entsprechend den hinteren, unteren Lungenlappen, leeren Schall an circumscripten Stellen (Atelektase?). — Der Husten zuerst spärlich und trocken, fördert später, wenn er häufiger und in Paroxysmen auftritt, zähes Sputum zu Tage.

Innerhalb zwölf bis vierundzwanzig Stunden mildert sich der Anfall,

doch ist das Rasseln noch viele Tage nach dem Anfall zu hören. — Zwischen den Anfällen besteht Kurzathmigkeit.

Aetiologisch ist in diesem Fall von Interesse, dass die Anfälle stets mit einem Schnupfen angefangen haben. Gewöhnlich brachte eines der Kinder den Schnupfen mit nach Hause und die anderen Kinder wurden auch davon ergriffen, wie man das ja so häufig in Familien sieht. Sobald die Reihe an unsere kleine Patientin kam, bekam sie auch beinahe regelmässig das ominöse Rasseln, aus welchem heraus sich dann der typische asthmatische Anfall entwickelte. Die Mutter, welche jede Einzelheit der Krankheit mit grosser Genauigkeit beobachtet, glaubt, dass es von grosser Wichtigkeit ist, prophylactisch unsere Patientin, von den anderen Kindern zu entfernen, sowie sich dieselben eine Erkältung zugezogen haben.

Wir haben es also hier mit einem rhachitischen Kinde zu thun, welches durch heftige Anfälle von Bronchospasmus, ein ausgebreitetes vesiculäres Emphysem acquirirte. Woher die Disposition, bleibt unaufgeklärt, Masern und Keuchhusten sind nicht vorausgegangen.

Das Emphysem hat sich, seitdem die Anfälle nicht mehr aufgetreten sind, wesentlich gebessert. Es ist nicht anzunehmen, dass sich organische Veränderungen am Herzen ausgebildet haben, und es ist für den Fall eine recht günstige Prognose zu stellen. —

Es liegt nicht in unserer Absicht auf eine detaillirte Besprechung differential-diagnostischer Punkte hier einzugehen. Prognostisch wichtig ist es, genau zwischen Asthma bronchiale und Bronchitis capillaris zu unterscheiden. Letztere ist eine der gefährlichsten Krankheiten des Kindesalters. Quoad vitam ist das Asthma aber prognostisch günstig. Stets soll man auch an Lungenoedem denken.

Therapeutisch nur einige kurze Bemerkungen. Gegen die Asthmaanfälle hat uns das Morphium, in dem zuletzt angeführten Fall gute Dienste geleistet. Ein Tropfen der officinellen Solutio Magendie schien den Anfall, wenn nicht geradezu zu couspiren, doch wesentlich zu erleichtern. Brom, Jod, Ammonium und schliesslich Digitalis thaten gute Dienste. Salpeterpapier, Räucherkerzchen und dergl. m. schienen auch günstig einzuwirken. Einmal probirten wir das von Frankreich aus empfohlene Pyridin *(Germain Sée)*, doch ohne eclatanten Erfolg.

Feuchte Witterung schien in unseren Fällen Erleichterung zu bringen. Im Sommer wirkt am günstigsten Seeluft ein. Seebäder wurden nicht erlaubt. — Die pneumatische Kammer erzielte auch vorübergehenden Erfolg.—Ein halbprocentiger Kochsalzspray hatte eine vorzügliche Wirkung im letzten Fall.

Fussend auf die, erst in der neuen Zeit erworbenen Kenntnisse über Nasen- und Rachenasthma, wird man diesen Organen besondere Aufmerksamkeit schenken müssen. *Soltmann* hat einen Fall von Asthma durch Entfernung von stark vergrösserten Tonsillen geheilt. Ob die Entfernung der Rachentonsille in unserem Fall günstig eingewirkt hat lässt sich ohne Weiteres nicht bestimmen. Dass auch die zugrunde-

liegenden Krankheiten (Rhachitis etc.) behandelt werden müssen braucht nur angedeutet zu werden.

Zum Schluss noch eine Bemerkung: Es gibt eine Form von Bronchitis bei Kindern, welche mit Giemen und Pfeifen namentlich während der Exspiration einhergeht. Die Eltern geben an, dass die Kinder von Zeit zu Zeit schwer athmen, ganz abgesehen von den Erscheinungen zu welchen der gewöhnliche Bronchialcatarrh an sich Anlass gibt. Es scheint als spielten bei dieser Form der Bronchitis spastische Momente geringeren Grades mit, als wie sie dem Bronchialasthma zukommen. Dieselben stellen gewissermassen die Uebergangsformen zwischen Bronchitis und Asthma dar. Diese Formen von Bronchitis hat *Politzer* im Sinn, wenn er von Bronchialasthma spricht, welches mit gleichzeitiger Bronchitis und Fieber einhergeht. Das Bronchialasthma wird in dieser Combination von der begleitenden Bronchitis leicht gänzlich verdeckt.—

Sollte es mir gelungen sein, die Aufmerksamkeit der Collegen auf die asthmatischen und emphysematösen Zustände im Kindesalter zu lenken, so wäre der Zweck dieser Zeilen erfüllt.

Meinem Freunde, Herrn Dr. I. Adler, bin ich zu Dank verpflichtet für die freundliche Ueberlassung der zwei zuletzt angeführten Fälle, welche ich zu gleicher Zeit mit ihm beobachtete.

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Writer compliments

SOME OBSERVATIONS

ON THE

TREATMENT OF DIPHTHERIA.

BY

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SOME OBSERVATIONS ON THE TREATMENT OF DIPHTHERIA.

BASED ON FORTY-THREE CASES WITH ONE DEATH.

MR. PRESIDENT AND FELLOW MEMBERS:*

FIRST, I wish to speak in condemnation of the practice so easily acquired, of calling every case of "sore throat" met with, DIPHTHERIA. Such practices should be left to quacks and irregular practitioners. It is not to be expected that this class of self-styled physicians will resist the temptation to establish, or add to their reputation, by piling up a long line of cases of this disease, so dreaded by the laity; but respectable regular physicians may, and should avoid it. An irregular practitioner, with whose career in a neighboring town I was somewhat familiar, thus treated all his cases of pharyngitis for years. When, in follicular disease, the little white patches were discernible, his medicines produced miraculous cures by causing their disappearance; when no membrane was apparent, it was because of the potency of his remedies; all were diphtheria; those with the "white patches" of the most malignant type. After a few years of continuously successful practice in curing these grave(?) cases, during which none of his patients had died (except from complications), diphtheria prevailed epidemically in the town. In nearly all of this gentlemen's cases the disease progressed rapidly to a fatal issue. One after another died. In many it extended to the larynx, its progress not even delayed by his magic medication. Then did he rise equal to the occasion, and boastingly declare that he had never lost a patient from the dread disease. Then he performed several tracheotomies; as the patients died the cause was loudly proclaimed as croup; those not tracheotomized died from "septicæmia"; but in all that man's practice, no patient of his has yet succumbed to diphtheria. One of the gravest dangers with which society is menaced, is that of the physician who "never lost a case." A lesser danger is that one whose every "sore throat" is "diphtheria."

The diagnosis in the majority of cases, is not difficult. In some it requires time. In case of uncertainty I would isolate the patient

* Read before the Hampden District Medical Society, at Springfield, Mass., Sept. 18, 1888.

and await progress before committing myself to a diagnosis. Follicular tonsillitis is the only disease with which diphtheria is likely to be confounded. The former begins abruptly; symptoms severe; temperature high (103); duration short. In the latter the commencement is gradual; symptoms less severe; lower range of temperature; albuminuria (insidious). Appearance of the membrane. In first, no fibrin; second, always fibrin. First, will not coalesce within 12–24 hours; in second, coalescence will invariably take place within that time. In first, can be easily rubbed off; in second, more force must be exercised, and this is followed by bleeding, etc. In diphtheria, in the majority of cases, there is but one centre, and from this the exudation extends to surrounding parts.

In April, 1887, I read before the Eastern Hampden Medical Association the following "Note on the Treatment of Diphtheria":

"I perhaps should apologize for bringing before you further suggestions regarding the treatment of this disorder. It is generally conceded that the results following the same treatment in different epidemics of this disease, being so variable, it is necessary that many cases of undoubted severity be successfully treated before the claim of a satisfactory method can be upheld. I have for years, as some of you are aware, endeavored to control the disease by rendering aseptic the local lesions, and at the same time adminstering remedies to prevent sysmetic infection. I at one time advocated the local use and internal administration of chlorine, as recommended by Cohen and used extensively by Calkins of this city. This was an excellent disinfectant and antiseptic, but experience has convinced me that if given indiscriminately, in all cases, there will quite too frequently appear a patient whose heart muscle will be weakened by its use to such an extent that death will ensue from such cardiac asthenia, even though the local lesions are improving. Cohen refers, also, to its tendency to produce renal irritation. We hear much of the heroic treatment of the disease, from extensive and thorough local cauterization, to the administration, in enormous doses, of nearly every remedy in the materia medica; and again see men stand indolently by, allowing their patients to die, while no attempt is made to resist the advance of the morbid processes. Both methods are equally unscientific and merit condemnation. For twelve years I have carefully watched the investigation going on in this department of medicine, and have experimented somewhat myself in the endeavor to find some drug of equal disinfecting power with chlorine, and devoid of its unpleasant and possibly dangerous effects. Some time ago Prof. A. Jacobi, of New York, advocated the internal use of the bichloride of mercury. Since the publication of his report of some sixty cases thus treated, personally and in consultation, I have not, until recently, attended a case in which the use of the drug has been dispensed with. Not discarding other medication, but also administering iron, potash, alcohol, the benzoates, etc., I have felt that in some of the cases the curative action was not so prompt as might be desired. One may form opinions regarding the action of medicines, if cases are carefully watched, without waiting for a fatal case to emphasize the slow action or total inefficiency of a remedy. Some two years since Letzerich published some observations regarding the curative action of benzoate of sodium in this disease. I have used it in a considerable prac-

tice, and although convinced of its general good effects, have not felt warranted in bringing it before you as of special worth in the treatment of diphtheria, until I had had opportunity to test it in some undoubtedly severe case. It was not my intention to exclude, in the treatment, other remedies whose action might be said to modify the effect of this drug. It was simply to so treat the patient as to most speedily conduce to recovery.

"Last week I had the long awaited opportunity in a case of malignant diphtheria. The patient, a girl aged five, previously healthy; pulse 188, temp. 104, face flushed and prolabia cyanotic, eyes suffused, mind sluggish, child lying in a partial stupor, vomiting. Examination showed tonsils, soft palate, uvula, and posterior wall of pharynx, so far as visible, covered with the peculiar dark greyish colored membrane. Respiration taking place almost entirely through the mouth, the post-nasal space being filled with the membranous formation. Excluding other children from the room, the treatment adopted was as follows: Every hour the child was given a dessert-spoonful of the following: sodium benzoate, dr.4; water, oz.7½; cinnamon water, oz.½. Thus taking 8 grains of the sodii salt hourly. With each dose a pill containing ¼gr. of calcium sulphide was given. I then prepared a solution of the sodii benzoate of the strength of seven drams to eight ounces of water, which was used in a hand atomizer every half hour, the solution being warmed before using. When the child was awake she was made to open her mouth as widely as possible during atomizations, but when asleep was not disturbed during its administration. The spray was thrown for about five minutes at each application. At the beginning, the child was given, every hour, a powder containing one-sixth gr. of the mild chloride of mercury, until six powders had been taken. No other treatment was pursued, the diet consisting of nothing but milk, which was given freely. As a precautionary measure (referring to other members of the family), and of possible beneficial action to the patient, I had a large open vessel placed upon the stove in the sick room, in which was constantly kept water sufficiently warm to produce steam. As often as necessary I directed that there should be poured into this vessel a dessertspoonful of the following: acid, carbolic; ol. turpentine; ol. eucalyptus, equal parts. The atmosphere of the room was thus constantly impregnated with these disinfectants. The day following my first visit the patient was in every way improved. The second day the membrane upon fauces had begun to curl up at the edges. The third day a large portion had been thrown off. No new appeared. The fourth day the membrane had disappeared, leaving a congested surface. From symptoms when the patient attempted breathing through the nostrils I concluded that there was still some membrane in the posterior nares, but could see none. Fifth day this trouble had disappeared, and the only difficulty complained of was some prostration, which seldom fails to succeed an attack of true diphtheria, although it was less in this case than might have been anticipated from its severity.

"I was led to adopt this treatment from the good result following its use in the hands of Dr. Brondel, reported in the Bull. Gén. de Therap. for Nov., 1886. My treatment was similar to that employed by him, with the exception that I did not feel warranted in dispensing with the mercurial at the commencement, for I am confident that by its use we can in every case shorten the duration of the disease. I have never seen the peculiar shrinking, curling appearance of the diphtheritic membrane, occurring so *early* in the disease, except in cases in which mercury has been adminstered. Dr,

Brondel reports 200 consecutive cases treated by the method outlined, without one death. In an editorial notice of the report, the Medical Record for January 8, 1887, says: 'Even excluding fifty per cent. of the cases upon the possibility of a mistaken diagnosis, he still has one hundred cases without a death.'

"Dr. B. uses tonics and antipyretics in conjunction with the remedy when they are indicated. I also should use them if it seemed necessary. In my own case, after the membrane had disappeared I gave full doses of iron (tr. chloride) to combat the prostration attendant upon the disease. I do not offer this as a routine medication in the treatment of diphtheria, as I do not favor routine in the practice of medicine. My suggestion is that this may form a basis for the treatment of these troublesome cases. The desired result is to be accomplished by attention to thorough antisepsis and disinfection locally, and prevention of "systemic" infection. So far as my information extends, this is at present the best means with which we are acquainted to attain these ends; modifying the treatment to meet special indications; using tonics, stimulants and antipyretics as may be indicated; particularly remembering that the drug which I have recommended is not to be given according to the dosage laid down in the books, where it is said that one to two drams may be given an adult daily. (In the case just cited, I administered hourly, for about one hundred consecutive hours, eight grains of the drug to a child five years of age, or nearly three and three-fourths drams daily.) Should give proportionally larger doses to adults. Should not decrease quantity proportionally for younger children, as they bear the drug better. Should give a quantity sufficient to prevent systemic infection if possible, and especially should I give the drug at short intervals as recommended. Thus adminstered there is little opportunity afforded for absorption of decomposing materials from the diseased surfaces.

"I wish to add, without going into detail regarding the various and convincing proofs of the beneficial effects of the medication adopted in the case just reported, that the progress from day to day, and the condition of the patient when the membrane had disappeared, was as satisfactory as any case which I have ever attended. It may be pertinent to add that the family were sufficiently anxious about the termination, to carry out directions minutely and intelligently. This may in part have been due to the fact that they had lost two children with the disease, under other treatment. I was struck with the persistency of their inquiries as to whether they should waken the child to administer the medicines, and they gave as a reason for this questioning, the fact that they had been admonished in the other cases, that if the children slept soundly, they were more harmed than benefitted by being roused out of refreshing sleep to take medicine. Also they had been told that if the children struggled greatly they need not insist upon the medicine being taken, as if they were thus allowed to exert themselves it would produce exhaustion more than compensating for the benefit accruing from medication. I informed the parents that my own convictions were opposed to those advanced, and, believing that the prostration and weak heart so often seen in diphtheria was due to the entrance into the systemic circulation of poisonous material, and if such entrance of septic matter could be prevented, exhaustion would not occur; therefore, I insisted that the child be awakened every hour, night and day, and the drugs administered. I directed that if the child struggled and refused to swallow, two persons administer it, one gently but firmly restraining her, the other giving the medicine. I predicted that after a few ineffectual attempts at resistance

the child would take the drugs offered without trouble. True enough, after giving it as above outlined three or four times, the child asked to be allowed to drink it from a glass, and continued to do so until well, and informed me at my next visit that the medicine was "good," asking that it might be given oftener. This much for the policy of allowing a sick child to be deprived of the benefits of medication because of morbid sentiment or weakness. It being admitted that the majority of deaths in diphtheria are due to systemic infection, or extension of the local deposition of membrane, it seems incredible that any conscientious physician can calmly stand by and allow his patient to die, when intelligence in selection of remedies and firmness in their administration will almost invariably effect a cure. No medication will succeed in this disease, unless the administration be thorough. It is little less than manslaughter to allow a patient to die without an effort being made to resist the progress of the disease."

The week following the reading of the foregoing remarks, I was called to attend a child aged 14 months, in the same family in which had occurred the case reported. Found slight swelling of the tonsils and adjacent lymphatics; a thin whitish membrane covering both tonsils; temperature 102; pulse 143; respiration slightly accelerated; face flushed; tongue covered with a light fur; had vomited once. Made diagnosis of diphtheria, and ordered treatment similar to that pursued in other case. Next day, feeling indisposed, I requested a physician from an adjoining town to see the case for me, which he kindly did. Calling at my house upon his return, he reported that no membrane was visible upon the fauces; child bright and apparently convalescent. He expressed the opinion that the case was one of pharyngitis, so often seen during the prevalence of diphtheria, and would probably recover without further trouble. As a precautionary measure he had directed that the medicines be continued until the following day. I then saw the patient and found the fauces normal in appearance; temperature normal; pulse 110; respiration 24. Child appeared well and took nourishment freely, and apparently as when in health. Directing that the remedies be continued in gradually decreasing doses for another day, I dismissed the case, concurring in the opinion that a mistake in diagnosis had been made, and that I had to do with a simple pharyngitis. On the eighth day following I was again called to see the child, the parents stating that for the past two nights there seemed to be some nasal obstruction which prevented the child sleeping with closed mouth. During last few hours this interference with respiration had increased to such an extent that the child kept the mouth open continuously, both when awake and when asleep. Hastening to the house I found the diphtheritic membrane completely filling the nostrils and protruding anteriorly. Examination of the throat also disclosed membrane on post-pharyngeal walls, uvula and covering posterior third of tonsils. The medication before employed was again brought into use; the spray being forced as far as possible into the anterior nares, in addition to the other methods. The following day there was sufficient retraction or

shrinking of the membrane occluding the nostrils to permit the spray being forced through into the post-nasal space. The membrane rapidly disappeared, and after four days none was perceptible, either in the throat or nose. The child rapidly regained strength, and after two days further treatment was again discharged. I believe this case was, at the beginning, one of diphtheritic affection, and that the thorough use of the drugs at the commencement caused the membrane to disappear from all parts exposed to view, but that a small foci somewhere in post-nasal space (possibly upon posterior surface of soft palate) was left after the first attack, which upon the discontinuance of the remedies, had gradually extended until the post-nasal space and both nostrils were completely filled. This illustrates two matters of importance. First, that in cases where the disease is seemingly aborted, no examination can be made in a young child sufficiently thorough to determine whether or not the mucous surfaces are entirely free from the presence of false membrane; and second, that in a case seen early, and in which the morbid process is apparently arrested, the continuance of the remedies for two or three days longer is not sufficient in every case to thoroughly eradicate the disease. It will of course depend upon whether the remaining nidus of morbid material is located in a situation where it is exposed to the full action of the administered drugs, or whether, lying in some inaccessible region, where the remedy fails to come in contact with it at all, or if at all, does so incompletely. It may be that such a condition would happen but rarely, but bearing in mind the possibility of this occurrence, it is well to continue the remedies for several days, or keep the patient under close observation after discontinuing their use, for a sufficient time to guard against extension of the membrane to the degree which occurred in the case reported.

After the recovery of this case, the house (including cellar) was again thoroughly disinfected by the fumes of sulphurous acid. Neither the two remaining children, nor others in the house using the same halls and cellar, have since contracted the disease. This child was probably infected from the sister first sick, through the medium of the mother, who attended the elder child throughout her illness, and declined to cease nursing the latter patient during the meantime.

I would state that by combining glycerine in the proportion of 1oz. to the 8oz. spray solution, its efficiency is apparently increased, the glycerine enabling it to the more readily become deposited and diffused upon the mucous and pseudo-membrane; it also remains longer in contact therewith. One effect of the benzoate of soda, which I find is not commonly borne in mind by physicians (if generally known), is a peculiar exhilaration or excitement bordering, in some cases, on active delirium. This condition usually appears in those severe cases where it is found necessary to push the remedy in full doses for several consecutive days. I am quite well satisfied that such intoxication is more apt to occur and prove troublesome, in

cases where, for some reason, it seems inadvisable to begin the treatment by the administration of mercury. I have never observed these symptoms, save in a very mild degree, where the mercurial has been given at the outset. Whether this preliminary hepatic stimulation and unloading of the digestive tract; thus promoting increased activity of the nutritive functions; or the possible modification of the uric acid formation or elimination is sufficient to explain the better toleration by the system of the benzoates, I am unprepared to state. I simply state the facts as I have observed them, leaving the explanation for future study. The continued administration of mercury throughout the disease, is in many cases harmful.

Now a few words as to the theory upon which the treatment outlined, is based. First, I wish to state my belief in the duality of the diseases known as pseudo-membranous laryngitis, and diphtheria. Among my reasons for believing in their non-indentity are the following. The presumable non-contagiousness of membranous croup; I have never seen the disease contracted by a second member of a family, though no measures of isolation have been exercised, while it is quite common for several members of a family to contract diphtheria. In membranous croup death occurs from apnœa, in diphtheria from asthenia. After tracheotomy in croup the edges of the wound are not covered by an extension of the membranous material from within. In diphtheria the edges of the wound are usually quickly invaded. In the former disease there is little or no tendency to hemorrhage; in diphtheria such tendency is common.

The paralyses so commonly following diphtheria are not commonly seen after recovery from membranous croup. The lymphatics are not ordinarily involved in membranous croup, while the opposite is true as regards diphtheria. Lastly, I am not aware of the recording of a case in which the membranous exudation has been communicated to an external wound in case of membranous croup, while such acccident is not uncommon in diphtheria.

As regards the local or constitutional origin of the disease, I think it unquestionable that the preponderance of evidence is in favor of the former. Although such be the case, and the disease *is* primarily local, nearly every case becomes constitutional before being seen by the physician. When seen, therefore, we have a constitutional disease with which to deal, and such constitutional state may be continued and increased by auto-inoculation.

To return to the theory upon which the treatment recommended is based. Bearing in mind the indications to be met, in successfully combating this disease, viz.: to render aseptic the local membranous surfaces; prevent systemic infection, and last, if possible, prevent extension of the local disease to adjacent tissues, and to support the patient; we find the first indication met by our frequently repeated spraying of affected surfaces, also very likely to some extent by the continuous inhalations of the powerful germicides con-

stantly impregnating the atmosphere as they are carried in the steam generated as described.* The second indication is met by the administration of the calcium sulphide, as also very possibly from the benzoate of soda taken internally, and also absorbed from the mucous surfaces; the steam inhalations may also contribute to the same end. The third indication is met by the spray of soda, which I am now in the habit of reinforcing when necessary with a solution of equal parts of a 15 vol. solution of peroxide of hydrogen and glycerine. This is the most powerful, non-irritating astringent disinfectant with which I am acquainted. By its astringent effect upon the tissues surrounding the local deposit, it seems to effectually prevent any extension whatever of the false membrane. My practice has been to use the benzoated spray every half hour from the first visit; if at any time I note any tendency to extension of the deposit, I direct that the hydrogen per-oxide shall be sprayed thoroughly over the surfaces every three hours, still continuing the benzoated spray as before. In no case when so used have I seen any extension of the disease. In less than one-half the cases do I resort to it at all. If there is evidence that septic absorption is progressing from beneath the pseudo-membrane, attempts should be made to dissolve this protective shield by papain or trypsin. If there is any tendency to hemorrhage from the affected membrane or adjacent parts, it is instantly arrested by the peroxide spray. (Incidentally, I will state that this has also been of great service to me in epistaxis, several severe cases, idiopathic and following operations upon the nasal passages, having been instantly controlled by its use).

The peroxide, as you are aware, is a most potent antiseptic, deodorizer and disinfectant. Its use, therefore, in diphtheria is peculiarly beneficial. It decomposes at temperatures above 65 F., but if kept below that point retains its properties apparently indefinitely. I have some which was purchased nearly two years since, and is now as efficient and active as ever. The patient is supported by the free administration of liquids, preferably milk. The conclusions arrived at, as stated in the paper read in 1887, have been sustained by continued experience. One statement made by me at that time I wish to correct. That is in relation to the use of

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* When the odor from the steam inhalations produced as above described, is particularly offensive to the patient or the attendants, I have latterly used in its stead a vapor produced by burning a candle composed of Iodine 1dr., Acid Salicylic ½dr., Paraffine 2½ oz., the same having been prepared for me according to my directions, by J. H. Losee, Pharmacist, this city. The vapor thus produced is nearly odorless, and so far as I am able to determine is as effectual a disinfectant as the liquid combination described. Further, it is a most efficient deodorizer. As you will remember, the disinfectants in the *liquid* consist of carbolic acid, turpentine and eucalyptus; while in the vapor from the candle, they are iodine and carbolic acid (the latter from the decomposition of the salicylic acid). If it should be shown that the diphtheritic bacilli are more susceptible to the toxic influence of one or other of these substances, that would govern the choice.

antipyretics. I said in the former paper that I should approve of the use of antipyretics in certain conditions. It seems impossible for me to conceive of any condition, liable to occur in the course of a case of diphtheria, in which their use would be warranted. I have never resorted to them in this disease, but in hastily thinking of the matter while writing the first paper, it was my impression that cases might sometimes occur in which their use would be of benefit. Further, and more mature consideration of the subject, convinces me that such a position is untenable. With the vast amount of literature at hand concerning their use in this and other diseases, all tending to impress the fact that any remedy which is sufficiently potent to quickly lower the temperature, invariably tends to depress the heart, together with personal experience in other diseases which coincides with this opinion ; and remembering the tendency to heart failure in diphtheria, I repeat, that in my opinion, one is not justified in administering antipyretics in this disease. This much without regard to the, as yet, but theoretical supposition, of pyresis being, in many instances, a conservative process. I have said little of the treatment usually adopted by those writers with whom we are most familiar, partly because I felt that all would be cognizant of their methods, and partly because I believe their treatment to be less practicable than that here recommended.

I am aware of the method of administering teaspoonful doses every half hour, of the tincture of the chloride of iron, as recommended by some of our New York brethren, and have no doubt good results might follow from the ingestion of the contained alcohol, as well as from the astringent and antiseptic action of the medicament locally. But as a matter of fact, it is very difficult to induce a child to swallow and *retain* such doses of iron. I have attempted it, and find that it is many times vomited soon after being taken. This interferes with nourishment, and produces exhaustion. In some cases, it is said the iron will be retained if its use be persisted in, when vomiting follows the first few doses. The method of Jacobi, to which I alluded, of giving bichloride of mercury freely, as also alcohol, etc., has been re-inforced, as recommended by him latterly, by local douching of the parts with antiseptics by means of the syringe ; or if ear symptoms be present, he recommends that the patient be seated upright, with head inclined slightly backward, the physician or nurse being behind the patient, when, the head being firmly held with one hand, the other, holding a spoonful of the antiseptic, is carried to the nostril, and the solution slowly poured through into the posterior fauces. The use of the solution by either method is attended with considerable more difficulty than that suggested in this paper, while it has the disadvantage of requiring the personal attendance of the physician or a specially trained nurse at each douching ; otherwise the nasal cavities will fail to be thoroughly cleansed, and bungling attempts to control a struggling child will cause unnecessary ex-

haustion. Prof. Bartholow advises sulphur, lime water, lactic acid locally, as also inhalations of steam from fresh lime upon which water is poured. He prevents systemic infection by the use of alcohol and iodine internally, and uses quinia. Dr. J. Silas Cohen uses the chlorine water, before alluded to, as also disinfectant sprays in the sick room, and administers alcohol internally. He advises that pieces of ice be kept in the mouth, renewed as occasion demands, and gives carbonate of ammonia if failure of the vital powers threatens. He advises local applications of tincture chloride of iron, by means of a swab, to the local pseudo-membrane. Liebermeister advises, and seems to rely almost solely upon the local application to the diseased surfaces of sulphur in powder. This probably acts by the sulphur undergoing oxidation and liberating sulphurous acid. He keeps the patient in an atmosphere saturated with antiseptic sprays, administers nourishment and stimulants freely. Application of ice to the neck, externally, is also recommended. Camphor, ammonia and quinia are used to sustain failing powers.

These various methods are mentioned to illustrate that the object of treatment everywhere recognized as of primary and vital importance, is the local cleansing and disinfection of the affected surfaces ; prevention of systemic infection, and support of the patient. The more lowered the nutrition, the more rapidly does the pseudo-membrane extend. If the indications mentioned can be fully met, the disease is rendered mild and innocuous. In my opinion they *are* met by a pursuance of the course as marked out in this paper. The illustrious Sydenham says, "True medicine consists in the discovery of the real indications, rather than in the excogitation of remedies." I am not prepared to state that the benzoate of soda is a specific for the cure of diphtheria. It may even be contended that the local action of the drug is no different than would result from a thorough cleansing, at frequent intervals, by pure water alone. This I am not prepared to admit, and as to the value of the benzoates in preventing systemic infection, it is recognized by Bartholow, who, in writing of "Ulcerative Endocarditis," says, "As septic materials are circulating through the blood, the benzoate of ammonium or salicylic acid should be administered freely."—(Page 263, Practice of Medicine.) The peculiar toxic effect following its use in large doses, continued for a considerable time, shows that the remedy is not inert. As in every case in my own practice, as also in the practice of others with which I am familiar, where these toxic symptoms have been noted, recovery has taken place, even though the disease may have been of a more than usually severe type, it leads to the inference that the drug may possess some specific properties. Suspecting that the symptoms mentioned might be due to the action of the soda compound upon the renal secretion, the urine was carefully examined in the last two cases in which such phenomena were present. Nothing was discovered tending to support that theory.

If anything, there seemed less albumin than is frequently found during the progress of diphtheria. I have said nothing of the heroic treatment sometimes advocated, as the administration of 40 grain doses of calomel at frequent intervals ; swabbing of the affected surfaces freely with strong solutions of nitrate of silver; saturation of the tissues of the body with alcohol, etc. Regarding this latter method, which seems very popular in some quarters, it appears to commend itself to many because it saves all thought, and is so easily administered. I am reminded of the practitioner I met last winter in one of the large western cities, who, on being asked what he considered the best treatment for diphtheria, said: "As soon as I see the case I order a half tumbler each, of whiskey and glycerine, repeated every hour until the patient is drunk, then keep him so until recovery or death ensues." I inquired if the mortality from diphtheria was large? to which he replied, "Yes, more than fifty per cent. of those affected die." Considering the treatment, what other result could be anticipated? Still, there are physicians in this State, who, to-day, pursue practically the same methods. Doubtless all of them consider diphtheria a most dangerous disease.

The abuse of calomel or alcohol in the manner described, cannot be too severely condemned. The physician should not *add* danger, to that already existing. The caustic applications to the faucial mucous membrane have, I think, been proven of no more utility than other less severe measures. Alcoholic stimulants should be used in this disease, in precisely the same manner as in all other diseases; viz., whenever indicated by a failure of the vital powers, as shown by a frequent, feeble or intermittent pulse. Its routine administration in all cases, from the beginning to the termination of the disease, is, I believe, more injurious than beneficial. In conclusion, I would recall your attention to the fact that the treatment, as herein laid down, meets the indications as recognized by all authorities. The sulphides prevent systemic infection; the benzoated soda, whether as a simple cleansing agent or otherwise, renders the affected surface aseptic, and limits or prevents extension of the pseudo-membrane, both in depth and superficially. When this is not sufficient alone, the use of the per-oxide of hydrogen re-inforces its action; the sprays and the constant inhalations of steam with which the atmosphere is impregnated, carrying also powerful disinfectants, prevent extension into the larynx as elsewhere; the internal administration of the soda compound has, I believe, a powerful action in preventing the asthenic condition so often present. The laxative at the beginning unloads the primæ viæ, and places the digestive apparatus in the best possible condition to allow of the carrying of the nutritive processes to the highest point. All of the remedies (if the calcium sulphide is administered in pill form, or with very young children the pill crushed, and given in some thick substance, as jelly), are nearly or quite tasteless; the only taste of the soda

mixture being of the cinnamon water, which may be changed for any other flavored water if cinnamon is not liked. Thus one of the formidable difficulties attending medication in children is overcome. Using a hand atomizer, with a soft rubber tube attached, as here shown, the nostrils, post-nasal space, and entire throat can be kept perfectly clean without the necessity of raising the child from the pillow, and many times without awakening it; and last, everything connected with the diseased surfaces being so thoroughly disinfected from the outset, other members of the family are less liable to contract the disease. Of course it is not necessary to refer to what every intelligent practitioner is in the habit of doing, in this as every infectious disorder, viz., insisting that the clothing, bedding and excretions should be immediately disinfected as removed. On the recovery of the patient, the room (preferably the entire house) should be thoroughly disinfected by sulphurous acid gas, or other efficient means. If complete isolation of the younger members of the family has been enforced, seldom will it happen that more than one case of the disease will occur in the same family. The treatment advocated possesses all the elements of good which may be claimed for other methods, while devoid of the dangers attending the use of some of them, and the disagreeable features of nearly all of them. The faithful employment of the treatment does not prevent a resort to alcoholic stimulants, the iron preparations, etc., should the same be indicated; and both from theoretical and practical considerations I am led to believe that if the treatment is conscientiously carried out there will be few cases of death from diphtheria, and those cases will be such as have insidiously progressed until fatal systemic infection has taken place before the disease is recognized.

One word in closing. I hope the matter has been made sufficiently plain that no one will look upon this as a specific "benzoate of soda treatment," as has been done; with the result that all attention has been directed to the soda compound, and other indications remained unfulfilled. It will not suffice to render the surface of the pseudo-membrane aseptic by frequent cleansing, while from the deeper parts septic infection is allowed to occur. Neither will it be of benefit to endeavor to prevent such septic infection by administration of calcium sulphide, etc., and allow the poisonous materials to accumulate in the post-naso-pharyngeal cavities. Understanding just what it is wished to accomplish, the remedies are intelligently administered. When any indication fails to be met, it is known which drug needs re-inforcement. In such manner, and such only, can this, and other serious disease, be satisfactorily treated. I thank you for your attention.

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DEN GANG DER TEMPERATUR

BEI

MENINGITIS TUBERCULOSA BASILARIS DER KINDER

UND

DAS VERHALTEN DER PULSFREQUENZ ZU DERSELBEN.

INAUGURAL-DISSERTATION

ZUR ERLANGUNG

DER MEDICINISCHEN DOCTORWÜRDE

AN DER

GROSSHERZOGLICH BADISCHEN RUPRECHT-KAROLINISCHEN UNIVERSITÄT

IN HEIDELBERG.

VON

SIEGBERT BALABAN.

VON DER MEDICINISCHEN FACULTÄT ZUM
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DECAN:
HERR GEH. RATH PROF. DR. W. KÜHNE.

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CARL WINTER'S UNIVERSITÄTSBUCHHANDLUNG IN HEIDELBERG.
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CARL WINTER'S UNIVERSITÄTSBUCHHANDLUNG IN HEIDELBERG.
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VOM

VERFASSER.

Obſchon das Leiden, deſſen Temperaturgang ich unterſuchen will, ſchon einem HIPPOCRATES[1]) nicht gänzlich unbekannt war, ſo gebührt doch dem engliſchen Arzte ROBERT WHYTT unſtreitig das Verdienſt, zuerſt eine treue und ſorgfältige Beſchreibung des Krankheitsbildes gegeben zu haben. Durch ſeine in Edinburgh im Jahre 1768 erſchienene Abhandlung «Observations on the dropsy of the brain» wurde die Aufmerkſamkeit der ärztlichen Welt auf dieſe bis dahin als beſondere Affection der Meningen nicht genügend gewürdigte Krankheitsform hingelenkt. Seit ihm hat man ſich auch daran gewöhnt, bei der Meningitis tuberculosa einen Erguß in die Ventrikel anzunehmen, und mit GUERSANT[2]) und PAPAVOINE[3]), die die tuberkulöſe Natur des Leidens zuerſt erkannten, dann mit PARENT, MARTINET, GERHARD, RUFZ, RILLIET und BARTHEZ das Weſen derſelben in einer tuberkulöſen Erkrankung der Meningen der Gehirnbaſis zu ſuchen. RILLIET und BARTHEZ[4]), die Erſten, welche die Meningitis tuberculosa von der Meningitis simplex ſcharf trennten, deren Sitz meiſt die Oberfläche der Hemiſphären iſt, nahmen als die charakteriſtiſchſten pathologiſch-anatomiſchen Unterſcheidungsmerkmale der tuberkulöſen Form den Sitz derſelben an der Baſis und den Erguß in die Hirnhöhlen an.

Seit dem Erſcheinen von ROBERT WHYTT's Abhandlung iſt nun zwar die Meningitis tuberculosa der Gegenſtand zahlreicher Arbeiten geweſen, ſo daß in unſerer Zeit dieſelbe in pathologiſch-anatomiſcher, ſowie in kliniſcher Beziehung als wohl erforſcht betrachtet werden darf, in den meiſten, auch ſehr vollſtändigen Abhandlungen iſt jedoch die Temperatur faſt gänzlich unberückſichtigt geblieben, während der Puls weit größere Beachtung fand. So auffällig dies ſcheinen mag, ſo erklärlich iſt dieſe Nichtberückſichtigung der Temperatur in dieſem Leiden. Denn der Beginn der Temperaturſteigerung entzieht ſich ſehr oft der Beobachtung,

[1]) Cfr. H. de morbis L. II. Cap. VI. Edit. CHARTIER. T. VII. pag. 556.
[2]) Article Méningite in Dictionnaire. 1827. [3]) Journal hebdomadaire. 1830.
[4]) Traite clinique et pratique des maladies des enfants. Paris 1843.

entweder weil in den erſten Tagen des ſchleichenden Anfangs der Krankheit keine Beobachtung gemacht wird, oder weil das häufige Vorhandenſein von Complicationen bereits die Temperatur beeinfluſſen. Dieſe Thatſachen erklären uns einerſeits die negativen Reſultate einer Anzahl von Unterſuchungen und andererſeits die abweichenden Anſichten verſchiedener Beobachter bezüglich des Temperaturganges bei der tuberkulöſen Meningitis. Ich hoffe nun durch Zuſammenſtellen einer Reihe von Fällen aus der Literatur, ſowie durch eine Anzahl von Fällen aus der Heidelberger Kinder- und Poliklinik, die mir Herr Hofrath Profeſſor v. Dusch gütigſt überlaſſen hat und über welche genaue thermometriſche Angaben vorhanden ſind, zu einem poſitiven Reſultate zu gelangen, wenn ich auch von vornherein überzeugt bin, daß es ſchwer halten wird, im Gange der Temperatur eine Geſetzmäßigkeit zu entdecken. Ich will nur noch einige kurze hiſtoriſche Erörterungen vorausſchicken.

Vor den ſechziger Jahren hatten wir noch keine genauen Temperaturmeſſungen bei Meningitis tuberculosa, erſt ſeit dieſem Jahrzehnt beſitzen wir exactere Meſſungen über dieſelbe. Die älteren Beobachter, ſo auch Robert Whytt[1]), machen zwar einige Angaben über das Fieber, da ſie indeſſen keine Unterſuchung mit dem Thermometer machten, vielmehr ihre Angaben auf ſehr unſicheren Unterſuchungen beruhten, wie auf Schätzung der Wärme mittelſt der Hand, auf Vorhandenſein gerötheter Hautſtellen oder auf dem Puls, ſo kann von einer exacten Meſſung nicht die Rede ſein.

Die ſpäteren Autoren beſchäftigten ſich hauptſächlich mit dem pathologiſch-anatomiſchen Befund und legten das Hauptgewicht auf die entzündliche Congeſtion der Meningen, wie Quin[2]), Gölis[3]), Brachet[4]), Senn[5]).

In ſeiner Abhandlung über Hydrocephalus wird etwas ſpäter von Itard[6]) das Fieber erwähnt, und iſt es nicht ohne Intereſſe, daß ſchon er demſelben jede Charakteriſtik abſprach.

Darauf folgt Henke[7]), der eine erſte febrile Periode, eine mittlere fieberfreie und eine Endperiode hervorhebt; in der Endperiode erreicht nach ihm das Fieber die höchsten Grade.

Fabre und Constant[8]) conſtatiren unregelmäßige Fieberanfälle, beſchäftigen ſich aber, ſowie Rufz[9]), Papavoine[10]) und andere Autoren dieſer Zeit hauptſächlich mit der Frage der tuberkulöſen Natur der Krankheit. Die zahlreichen Arbeiten dieſer Zeit laſſen die Temperatur meiſt unberückſichtigt, oder dieſelbe bildet wenigſtens

[1]) Op. cit. [2]) A Treatise on the dropsy of the brain. London 1790. [3]) Praktiſche Abhandlungen über die vorzüglichſten Krankheiten des kindlichen Alters. Wien 1815. [4]) Essai sur l'hydrocéphalie ou hydrop. aigue. Paris 1818. [5]) Recherches anatomiques-pathologiques sur la Méningite aigue. Paris 1823. [6]) Dictionnaire des Sciences médicales. 1818. [7]) Kinderkrankheiten. Frankfurt 1818. [8]) Mémoire inédit. 1835. [9]) Thèse de Paris. 1835. [10]) Op. cit.

nie den Gegenſtand ſorgſamer Betrachtung, bis wir mit dem Jahre 1843 wieder etwas genauere Angaben bei RILLIET und BARTHEZ[1]) über das Fieber finden. Von dieſen berühmten Autoren wird das Fieber im Prodromalſtadium geleugnet, es beginnt nach ihnen erſt mit dem wirklichen Anfang der Krankheit, manchmal mit leichten Schüttelfröſten, die höchſten Fiebergrade werden am Ende beobachtet.

Jetzt tritt eine Pauſe von faſt einem Vierteljahrhundert in der Beobachtung des Fiebers ein, wir finden keine nennenswerthen Angaben während dieſer Zeit über das Fieber vor; die Fieberreſultate beruhen wieder meiſt auf Pulsunterſuchung, und wir kommen erſt zu beſtimmteren Ergebniſſen mit der Einführung der thermometriſchen Exploration am Krankenbett.

WUNDERLICH[2]) beſpricht in ſeinem klaſſiſchen Werke den Fieberverlauf bei der Meningitis tuberculosa ungefähr wie folgt: Da die Krankheit ſchleichend beginnt, ſo entzieht ſich der Beginn der Temperaturerhöhung der Beobachtung entweder dadurch, weil eben in dieſer Zeit keine Beobachtung gemacht wird, oder weil vorhandene andere Störungen bereits Temperaturſteigerung bedingten. Während des Verlaufs wechſeln Exacerbationen mit Remiſſionen ab, zuweilen trete mehrtägige Apyrexie ein, gewöhnlich ſei der Typus remittirend. Sub finem vitae ſteige die Temperatur nur ausnahmsweiſe, gewöhnlich ſinke ſie, vorher febril, faſt bis zur Norm. In der Agonie ſinke ſie entweder fort oder es tritt noch vor dem Tode eine bedeutende Steigerung ein. Der Puls dagegen nehme an Frequenz immer zu, bis faſt zum Moment, in welchem das Herz ſeine Functionen einſtellt.

Erwähnenswerth iſt die Anſicht von VOGEL[3]), nach ihm verläuft eine nur auf die Meningen beſchränkte Tuberkuloſe überhaupt ohne Fieber.

Dagegen hebt ROGER[4]), geſtützt auf zahlreiche Unterſuchungen der Temperatur bei Kindern, eine große Unregelmäßigkeit im Temperaturgange der Meningitis tuberculosa hervor; indeſſen ſoll die Temperatur nach ihm meiſt keine ſehr hohen Grade zeigen.

STEINER[5]) weiſt auf das plötzliche Steigen der Temperatur bis auf 40,0 am Ende der Krankheit hin.

Derſelben Anſicht iſt HENNIG[6]), der im Beginn der Krankheit nur mäßiges Fieber ſah, während die letzten Tage ſehr hohes Fieber aufwieſen.

Nach GERHARD[7]) und BOUCHUT[8]) zeigt die Krankheit manchmal im Prodromalſtadium unregelmäßiges Fieber, etwas tiefer in

[1]) Op. cit. [2]) Das Verhalten der Eigenwärme in Krankheiten. Leipzig 1870. [3]) Lehrbuch der Kinderkrankheiten. Erlangen 1863. [4]) Recherches cliniques sur les maladies de l'enfance. Paris 1872. [5]) Compendium der Kinderkrankheiten. Leipzig 1872. [6]) Lehrbuch der Krankheiten des Kindes. Leipzig 1864. [7]) Lehrbuch der Kinderkrankheiten. Tübingen 1871. [8]) Traité pratique des maladies des nouveaux-nés. Paris 1867.

der Mitte der Krankheit, vor dem Ende trete eine bedeutende Temperaturſteigerung ein.

Archambault[1]) hebt hervor, daß ſchon das Prodromalſtadium Fieber aufweiſe, welches ſich durch Schnelligkeit des Pulſes und Erhöhung der Temperatur darſtellt, dasſelbe zeige continuirlich remittirenden Typus und werde immer ſtärker, je weiter das Prodromalſtadium vorrückt. Die Krankheit ſelbſt zeige am Anfange bedeutendere Grade, in der Mitte ſinke die Temperatur in etwas, wogegen das Ende die bedeutendſten Steigerungen aufweiſe.

In Bertalot's[2]) Abhandlung über Meningitis tuberculosa der Kinder iſt auch der Unregelmäßigkeit des Fiebers Erwähnung gethan. Der Beginn ſei entweder normal oder zeige nur mäßiges Fieber, mit intercurrenten Exacerbationen bis gegen 39,5, am Ende behält das Fieber dieſen Typus bei, oder es trete ein plötzliches Steigen oder Sinken ein.

In den Pariſer Thèsen von Leroux[3]) und Boutan[4]) erreicht die Temperatur während der Krankheit höhere Grade, auch ſchon das Prodromalſtadium zeige etwas Fieber, die höchſten Temperaturen beobachte man gegen das Lebensende.

Henoch[5]) conſtatirt, daß die Meningitis tuberculosa durchaus keine charakteriſtiſche Fieberkurve zeige, der Krankheitsverlauf ſei mit erheblichen Schwankungen der Temperatur verbunden, die Exacerbationen fallen faſt immer auf den Abend. Vor dem Tode trete in den meiſten Fällen eine rapide und ſehr hohe Temperaturſteigerung ein, nur ſelten ſinke dieſelbe auf 38—39 vor dem Tode. Die diesbezüglichen Fälle, aus welchen Henoch dieſen Schluß zieht, habe ich geſammelt und werde ſie in dieſer Arbeit verwerthen.

d'Espine und Picot[6]) beobachteten ſchon im Prodromalſtadium Fieber, in den letzten Tagen erhöht ſich die Temperatur auf 40,0 bis 40,5 und erreicht ihr Maximum 41,0—41,5 im Augenblick des Todes, nach welchem ſie noch einige Minuten zu ſteigen fortfährt.

Trousseau[7]) ſah die Krankheit mit Fröſteln beginnen, die Mitte der Krankheit verläuft nach ihm fieberfrei, das Ende zeichnet ſich durch Temperaturſteigerung aus.

Smith[8]) beobachtete während der Krankheit einen unregelmäßigen Fieberverlauf, vor dem Tode ſtieg die Temperatur zuweilen auf 106—108 F. = 41,1—42,2 C.

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[1]) Dictionnaire encyclopédique des Sciences médicales. Paris 1873. [2]) Jahrbuch für Kinderheilkunde. 1876. [3]) De la Méningite tuberculeuse de l'enfance. Paris 1877. [4]) Essai sur la température, le pouls et respiration dans la Méningite tuberculeuse des enfants. Paris 1877. [5]) Charité-Annalen. N. F. IV. Berlin 1879. [6]) Grundriß der Kinderkrankheiten. Deutſch von Ehrenhaus. Leipzig 1874. [7]) Clinique médicale. Paris 1877. [8]) Clinical studies of disease in Children. London 1876.

Turin[1]), der ſich auf 38 Fälle ſtützt, kommt zu dem Schluſſe einer Temperaturerhöhung in einem oder zwei Stadien, ſelten ſei die Temperatur während des ganzen Verlaufes erhöht. Der Temperaturgang ſei unregelmäßig mit gewöhnlich remittirendem Typus.

Aus dieſer kurzen hiſtoriſchen Zuſammenſtellung erſehen wir, wie verſchieden die Temperaturverhältniſſe bei Meningitis tuberculosa von den verſchiedenen Beobachtern dargeſtellt wurden. Ich will nun verſuchen, ob ich durch Anführen ſtatiſtiſcher Zahlen zu einem poſitiven Ergebniſſe gelangen werde, und theile ich meine Fälle in 3 Kategorien ein. I. Kategorie: Fälle, die am Todestage Normal — nahezu Normal — oder mäßig erhöte Temperatur aufweiſen, hierher rechne ich die Fälle von 37,0—38,5. II. Kategorie: Fälle, die am Todestage höhere oder ſehr hohe Temperaturen aufweiſen, die Fälle von 38,6 und darüber. III. Kategorie: Fälle, die am Todestage ſubnormale oder ſehr tiefe Temperatur zeigen.

I. Kategorie.

1. Fall. Franz Herrmann, 2 Jahre alt, wurde am 14. Mai 1873 wegen Coxitis in die Heidelberger Kinderklinik aufgenommen. Am 1. Dezbr. traten die erſten meningitiſchen Erſcheinungen auf.

Fiebertabelle.

		Mrgs.	Mttgs.	Abds.			
Am	1. Dezember Temp.	37,0	—	37,4	Puls	—	—
»	2. » »	37,9	—	38,4	»	—	—
»	3. » »	37,8	—	38,8	»	—	—
»	4. » »	37,8	38,5	37,9	»	108	—
»	5. » »	38,0	38,2	38,3	»	92	—
»	6. » »	37,8	38,2	38,2	»	—	—
»	7. » »	38,0	38,6	38,0	»	—	—
»	8. » »	38,4	38,6	38,2	»	100	—
»	9. » »	39,0	38,6	38,2	»	136	140
»	10. » »	38,8	39,2	39,4	»	140	—
»	11. » »	38,4	38,6	38,6	»	128	140
»	12. » »	37,8	38,2	38,2	»	80	—
»	13. » »	37,8	—	—	»	180	—

Section am 14. Dezember, 9 Uhr Vormittags.

Bei Eröffnung des Schädels entleert ſich viel Flüſſigkeit. An der Gehirnbaſis iſt die Pia zwiſchen dem Chiasma NN. opt. und Pons getrübt, im Subarachnoidalraum dünn gallertiges Exſudat. In der Sylviſchen Grube beiderſeits zahlreiche, hirſekorngroße Knötchen, beſonders im Verlauf der kleinen Gefäße. Ventrikel weit, enthalten copiöſe, klare Flüſſigkeit. Ependym beiderſeits mit zahlreichen Knötchen beſetzt.

[1]) Jahrbuch für Kinderheilkunde. Leipzig 1880.

Anatomiſche Diagnoſe. Meningitis basilaris tuberculosa, Bronchitis, Bronchopneumonie, Atelektaſe, Coxitis.

Der Temperaturverlauf iſt in dieſem Falle ſehr ſchwankend; am 10. bis auf 39,4 ſteigend, iſt die Temperatur an den folgenden Tagen niedriger, auch am Todestage erreicht dieſelbe nur 37,8. Meiſt ſind indeſſen abendliche mäßige Exacerbationen. Die Pulsfrequenz iſt am Todestage ſehr hoch, aber ſowohl an dieſem, wie auch während der anderen Tage ohne Coordination an die Temperatur.

2. Fall. Eliſe Müller, 1 Jahr alt, wurde am 26. Februar 1874 mit Eczema capitis et faciei im Heidelberger Kinderſpital aufgenommen. Am 20. April traten die erſten meningitiſchen Erſcheinungen auf.

Fiebertabelle.

		Mrgs.	Mttgs.	Abds.	
Den 22.	April T.	38,0	—	37,6	P. 138
» 23.	» »	38,4	37,6	37,2	» 138
» 24.	» »	37,2	37,6	37,4	» 140
» 25.	» »	38,4	37,2	37,0	» 160
» 26.	» »	37,6	37,2	38,0	» 132.

Tod ½12 Uhr Nachts.

Section am 27. April 12 Uhr Mittag. An der Gehirnbaſis zwiſchen Chiasma und Pons iſt die Arachnoidea und Pia im Zuſtande einer gallertigen, ſtellenweiſe gelblich getrübter Infiltration. Dieſe Infiltration reicht durch die ganzen Fossae Sylvii, wo ſich auch eine Anzahl ſtecknadelkopfgroßer, weißlicher Knötchen findet. Ebenſo Infiltration und ſehr zahlreiche Knötchen in der Spalte zwiſchen Groß- und Kleinhirn. Ventrikel ſtark erweitert und mit trüber Flüſſigkeit gefüllt. In den meiſten übrigen Organen ebenfalls pathologiſche Veränderungen.

Der Temperaturverlauf iſt äußerſt ſchwankend, am 22., 23. und 25. iſt Abends die Temperatur niedriger als am Morgen, dieſelbe erhebt ſich nicht über 38,4. Die Pulsfrequenz iſt hoch und nicht an die Temperatur coordinirt.

3. Fall. Georg Merkel, 4 Jahre alt, wurde am 2. Mai 1880 mit chroniſch käſiger Pneumonie im Heidelberger Kinderſpital aufgenommen. Eintritt meningitiſcher Erſcheinungen am 1. Juni 1882.

Fiebertabelle.

	Mrgs.	Abds.		Mrgs.	Abds.
Den 1. Juni T.	38,5	39,5	Den 8. Juni T.	37,1	38,7
» 2. » »	38,1	39,4	» 9. » »	37,3	39,5
» 3. » »	38,0	39,8	» 10. » »	38,0	39,7
» 4. » »	38,3	39,5	» 11. » »	38,1	38,6
» 5. » »	38,4	39,5	» 12. » »	37,5	39,5
» 6. » »	38,0	39,0	» 13. » »	37,6	39,0
» 7. » »	38,1	39,7	» 14. » »	37,4	38,4

	Mrgs.	Abds.		Mrgs.	Abds.
Den 15. Juni T.	38,2	38,4	Den 18. Juni T.	37,7	37,6
» 16. » »	37,3	37,6	» 19. » »	37,1	37,5
» 17. » »	36,9	37,2	» 20. » »	36,2	36,1.

Am 21. Juni 37,0. Geſtorben Nachts $^3/_4$12.

Autopſie. Dura mater enthält im Sinus friſches Gerinnſel. Pons ſehr ſtark ſulzig und eitrig infiltrirt, beſonders ſtark entſprechend den pialen Auskleidungen der Fossae Sylvii, die überdies von einer ganzen Unmaſſe grauer Knötchen durchſetzt ſind. Die Seitenventrikel ſind erweitert und mit klarer Flüſſigkeit gefüllt. Außerdem noch chroniſch ulceröſe Pneumonie, käſige Degeneration der bronchialen Lymphdrüſen, tuberkulöſe Peritonitis und ein circa nußgroßer, derber, käſiger Knoten in der rechten Kleinhirnhälfte.

Der Temperaturverlauf iſt in dieſem Falle ganz unregelmäßig, nur herrſchen, mit Ausnahme des 18. und 20. Juni, conſtante Exacerbationen vor. Während des ganzen Verlaufs ſchwankt die Temperatur Morgens zwiſchen 36,2 und 38,5, Abends zwiſchen 36,1 und 39,7. Am vorletzten Tage iſt die Temperatur ſowohl Morgens als Abends ſubnormal, am Todestage auch 37 nur erreichend.

4. Fall. Guſtav Behrens, $2^1/_2$ Jahre alt, wurde am 4. März 1874 mit bereits entwickelter Meningitis tuberculosa basilaris in die Heidelberger Kinderklinik aufgenommen.

Fiebertabelle.

	Mrgs.	Mttgs.	Abds.	
Den 4. März T.	37,8	37,0	37,2	P. 84
» 4. » »	—	10 Uhr Abds.	36,4	» —
» 5. » »	37,4	37,0	37,2	» 88
» 6. » »	37,2	—	37,0	» 130
» 7. » »	37,0	—	37,2	» 112
» 8. » »	38,0	—	—	über 200.

Unter ſtets zunehmender Ruhe und größerem Sopor iſt das Kind $^1/_2$9 Uhr Abends geſtorben.

Die poſtmortale Meſſung betrug 5 Minuten nach $^1/_2$9 38,0
9 Uhr 29 Minuten 38,0
10 » 37,4,
alle 10 M. weiter, 36,8—36,2—35,8—35,2.

Section am 10. März 12 Uhr Mittags. Die Windungen des Gehirns ſtark abgeflacht, die Maſchen der Arachnoides und Pia an der Baſis eitrig infiltrirt und von miliaren Knötchen durchſetzt. Dieſelbe Veränderung zeigt die piale Auskleidung der Sylviſchen Gruben. Die Seitenventrikel beträchtlich erweitert und mit Serum gefüllt. Außerdem noch chroniſche Bronchitis, Bronchektaſie und Verkäſung der Bronchialdrüſen.

Temperaturverlauf ſehr unregelmäßig, auch herrſchen conſtante abendliche Remiſſionen (mit Ausnahme des 7.) vor. Die Pulsfrequenz iſt ohne Spur von Coordination an die Temperatur.

5. Fall. Johanna Roos, $2^1/_2$ Jahr alt, wurde am 11. Februar 1880 mit entwickelter Meningitis und Miliartuberkuloſe in die Heidelberger Kinderklinik aufgenommen.

Fiebertabelle.

		Mrgs.	Mttgs.	Abds.		
Den 11.	Februar T.	—	—	38,4	P.	— —
» 12.	» »	39,0	39,1	39,6	»	104—108
» 13.	» »	38,6	37,2	38,4	»	— —
» 14.	» »	38,2	39,2	39,2	»	120—150
» 15.	» »	38,8	39,2	39,2	»	138—150
» 16.	» »	37,4	38,0	38,0	»	144— unzählbar.

Der Temperaturverlauf iſt höchſt unregelmäßig, am 12. 39,6 erreichend, zeigt die Temperatur an den folgenden Tagen bei weitem niedrigere Grade. Am 13. Mittags und am Morgen des Todestages iſt die Temperatur am niederſten. Indeſſen ſind (mit Ausnahme des 13.) conſtante Abendexacerbationen. Die Pulsfrequenz iſt eben ſowenig wie in den früheren Fällen der Temperatur coordinirt.

6. Fall. Käthchen Sommer, 10 Jahre alt, kam am 9. Juli 1883 in poliklinische Behandlung. Im 7. Jahre hat Patientin einen dreiwöchentlichen Typhus durchgemacht, auch wurde dieſelbe auf der Heidelberger chirurgiſchen Klinik wegen Skolio-Kyphoſe und einer Compreſſionsmyelitis behandelt. Seit 2 Tagen ſollen Kopfſchmerzen beſtehen, vorgeſtern und geſtern je einmal erbrochen.

Fiebertabelle.

Den 9.	Juli T.	Morgens u.	Abends	37,2	P.	60	ganz unregelmäßig.
» 10.	» »	»	»	normal	»	56, nach 3—4 Schlägen intermittirend.	
» 11.	» »	»	»	normal	»	80—120	
» 12.	» »	»	»	normal	»	134	
» 14.	» »	»	»	normal	»	—	
» 15.	» »	»	»	normal	»	80	
» 15.	» »	»	»	normal	»	130	

» 16. Die Angehörigen geben an, daß Patientin in der Nacht vom 15. auf den 16. geſtorben ſei.

Sectionsbefund. Die Piagefäße zeigen ſtarke venöſe Hyperämie, Pia läßt ſich leicht abziehen und zeigt in den Fossae Sylvii Trübung mit einigen grauen Knötchen durchſetzt. Gehirnwindungen etwas abgeflacht. In den Großhirnventrikeln findet ſich eine beträchtliche Menge einer klaren ſeröſen Flüſſigkeit, ſo daß die Seitenventrikel und das Corpus callosum ſtark geſpannt ſind. Außerdem Hypoſtaſe der linken Lunge, etwas Atelektaſe des mittleren rechten Lungenlappens, zum Theil Petrification der Bronchialdrüſen. An der Wirbelſäule iſt kein käſiger Herd zu finden.

Dieſer Fall zeichnet ſich dadurch aus, daß während des ganzen Verlaufs der Krankheit überhaupt die Temperatur ganz normal

blieb, welches Verhalten eben ſo ungeſetzmäßig iſt, wie das Verhalten der Temperatur in den vorhergehenden Fällen. Die Pulsfrequenz iſt ebenſo unregelmäßig und ohne Coordination an die Temperatur.

7. Fall. Paul Fitt, 1 Jahr 3 Monate alt, am 17. März 1869 im Kinderſpital aufgenommen, ſoll ſeit 2 Monaten krank ſein.

Fiebertabelle.	Mrgs.	Mttgs.	Abds.		Mrgs.	Mttgs.	Abds.
Den 17. März T.	—	38,0	38,0	P.	—	108	108
» 18. » »	37,7	38,3	37,7	»	68	100	72
» 19. » »	37,6	38,2	37,9	»	68	80	70
» 20. » »	37,7	38,5	38,2	»	86	120	100
» 21. » »	37,6	—	37,5	»	68	—	68.

Nach dem Tode im After gemeſſen: nach $^1/_4$ Stunde 37,8, von da ab jede $^1/_4$ Stunde weiter 35,8—33,8—33,4.

Section am 22. März Vormittags $11^1/_2$ Uhr.

Kopfhöhle: Dura mater mit dem Schädel verwachſen. Reichliches, gallertiges Exſudat zwiſchen Pia und Arachnoidea. Gyri beträchtlich abgeflacht. Sulziges Exſudat an der Basis cerebri von mäßiger Ausbreitung, mit reichlichen miliaren Tuberkeln. Reichlicher, ſeröſer Erguß in die Ventrikel. In der Chorioidea des rechten Auges ein grauer, ſtecknadelkopfgroßer Tuberkel, in den meiſten übrigen Organen pathologiſche Veränderungen.

Temperaturverlauf unregelmäßig, meiſt Normaltemperatur. Pulsfrequenz etwas mehr als in früheren Fällen der Temperatur coordinirt.

8. Fall. Ein Mädchen von 4 Jahren wird mit bereits vollſtändig entwickelter Meningitis tuberculosa basilaris im Kinderſpital aufgenommen.

Fiebertabelle.

Am 1. Tage T.	—	38,0	P.	—	102
» 2. » »	38,3	—37,9	»	118	120
» 3. » «	38,4	—38,3	»	126	114.

Am 4. Tage trat der Tod ein. Die poſtmortale Meſſung betrug $^1/_4$ Stunde nach dem Tode 38,3 und eine Stunde ſpäter 36,5.

Section 22 Stunden post mortem. Kopfhöhle: Schädeldach ziemlich dünn. Dura mater ſtark adhärent. Gehirnwindungen abgeflacht, Sulci verſtrichen. Grüngelbes eitriges Exſudat in den Maſchen der mit reichlichen grauen Knötchen verſehenen baſalen Pia. Beträchtlicher ſeröſer Erguß in den erweiterten Seitenventrikeln. Hochgradiges Oedem des Gehirns: Außerdem nur noch Verkäſung der Meſenterialdrüſen.

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[7]) Jahrbuch für Kinderheilkunde, Steffen, Leipzig 1869. Bd. II. p. 322

[8]) Steffen, Handbuch der Kinderkrankheiten. Bd. 5. II. pag. 475.

Temperaturverlauf ſchwankend, mit abendlichen mäßigen Remiſſionen. Der Puls iſt frequent und ohne Beiordnung an die Temperatur.

9. Fall. Ein 3 Jahre altes Kind, Namens Gleßmann, erkrankt am 15. Dezember 1874.

Fiebertabelle.	Mrgs.	Abds.		Mrgs.	Abds.
Den 18. Dezember T.	—	38,0	P.	—	88
» 19. » »	39,0	39,1	»	108	124
» 20. » »	38,0	38,7	»	176	164
» 21. » »	37,4	40,2	»	164	—
» 22. » »	38,5	?	»	132	—.

Krankheitsdauer 8 Tage. Temperatur ganz unregelmäßig, aber mit conſtanten Abendexacerbationen, Morgens zwiſchen 37,4 und 39,0, Abends zwiſchen 38,0 und 39,1 ſchwankend, am vorletzten Abend plötzlich 40,2 erreichend. Bemerkenswerth iſt in dieſem Falle das Sinken der Temperatur am Todestage auf 38,5, gleichzeitig Verminderung der Pulsfrequenz, ſonſt iſt die Pulsfrequenz jedoch nicht an die Temperatur beigeordnet.

10. Fall. Ein 3 Jahre altes Kind, Namens Klinger, aufgenommen am 27. Dezember 1875 mit den Symptomen einer rechtſeitigen Bronchopneumonie, zeigt am 20. März die erſten Symptome von Meningitis.

Fiebertabelle.	Mrgs.	Abds.		Mrgs.	Abds.
Den 20. März T.	37,8	38,5	P.	120	136
» 21. » »	38,3	38,4	»		
» 22. » »	38,0	39,6	»	108	120
» 23. » »	38,5	38,9	»		
» 24. » »	38,3	38,4	»		
» 25. » »	37,8	38,7	»	120	90
» 26. » »	38,7	38,1	»	100	95
» 27. » »	37,3	38,1	»	100	114
» 28. » »	38,0	38,1	»	164	.

Während des 9tägigen Verlaufs der Krankheit zeigt ſich die Temperatur äußerſt ſchwankend. Das einzige Conſtante iſt die wenn auch nur mäßige Erhebung in den Abendſtunden (mit Ausnahme des 26.). Aber von einem regelmäßigen Steigen und Fallen iſt keine Rede, am 3. Krankheitstage 39,6 erreichend, am Tage vor dem Tode Normaltemperatur. Pulsfrequenz ebenfalls unregelmäßig und ohne Beiordnung an die Temperatur.

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[9]) Henoch, Charité-Annalen, Bd. IV. pag. 507. Berlin 1879.
[10]) Henoch, Charité-Annalen, Bd. IV. pag. 507.

11. Fall. R. Maria, 2 Jahre alt, aufgenommen den 11. April 1875, ſoll ſeit 15 Tagen krank ſein.

Fiebertabelle.	Mrgs.	Abds.		Mrgs.	Abds.
Den 11. April T.	36,8	36,2	P.	100	160
» 12. » »	38,0	38,4	»	165	175
» 13. » »	36,4	36,6	»	140	160
» 14. » »	36,6	37,8	»	150	190
» 15. » »	36,2	—	»	140	155
» 16. » »	37,2	37,6	»	140	180.

Der Tod trat am folgenden Tage früh ein.

Die Autopſie ergab tuberkulöſe Granulationen an der Hirnbaſis, einen purulenten Erguß unter der Pia mater und beträchtliche Füllung der Ventrikel mit ſeröſer Flüſſigkeit.

Die Temperatur zeigt ſich ſchwankend, indeſſen ſind (mit Ausnahme des 11.) conſtante Abendexacerbationen, ſteigt nicht über 38,4, meiſtentheils iſt die Temperatur ſubnormal, während die Pulsfrequenz ſehr bedeutend iſt und ſich zwar den Temperaturſchwankungen (wieder mit Ausnahme des 11.) coordinirt, aber in gar keinem Größenverhältniſſe zu derſelben ſteht.

II. Kategorie.

12. Fall. Philipp Schneckenberger, 9 Monate alt, wurde am 19. October 1875 wegen Coxitis im Heidelberger Kinderſpital aufgenommen. Gegen den 11. November etwa zeigten ſich die erſten meningitiſchen Symptome.

Fiebertabelle.	Mrgs.	Abds.		Mrgs.	Abds.	
Den 11. Nov. T.	38,4	40,0	Den 22. Nov. T.	38,8	39,6	
» 12. » »	38,6	39,6	» 23. » »	39,0	39,8	
» 13. » »	38,2	40,0	» 24. » »	38,8	39,8	
» 14. » »	38,4	39,8	» 25. » »	39,2	39,6	
» 15. » »	38,2	40,2	» 26. » »	39,0	39,2	
» 16. » »	38,6	39,6	» 27. » »	38,8	39,0	
» 17. » »	38,8	39,8	» 28. » »	38,6	39,4	
» 18. » »	38,2	40,2	» 29. » »	38,8	37,6	P. 92—148
» 19. » »	39,4	39,8	» 30. » »	38,4	38,2	» 180—132
» 20. » »	38,8	39,8	» 1. Dezember T. Morgens 39,0,			
» 21. » »	39,2	39,8	Mittgs. 38,8, P. an der Carotis 160.			

Geſtorben um 12 Uhr Mittags. Die poſtmortale Meſſung ergab um ½ 1 38,2, dann weiteren Abfall.

Section am 4. Dezember 12 Uhr Mittags. Im Sinus der Dura mater viel Blut, Pia von mittlerem Blutgehalt, an der Convexität

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11) Boutan, op. cit. pag. 20.

ſtark durchfeuchtet. Gegen die Baſis hin geht dieſe Durchf in ein eitriges käſiges Exſudat über, welches in die Foss reicht und die Gegend zwiſchen Pons und Chiasma Die Ventrikel ſind mäßig erweitert und mit klarer F gefüllt. Außerdem allgemeine Miliartuberkuloſe und käſ Kniegelenkentzündung.

Der Temperaturverlauf iſt äußerſt unregelmäßig, am 15. und 18. erreicht die Temperatur die höchſte Ziffer 40, an den anderen Tagen (mit Ausnahme der beiden vorletzt ſchwankt ſie Morgens zwiſchen 38,2 und 39,4, Abends 39,0 und 39,8. Während nun die Temperatur während d Krankheitsverlaufs hohe Grade zeigt, zeichnen ſich die be letzten Tage durch mäßiges Fieber und Normaltempera ebenſo ſinkt die Temperatur direct vor dem Tode auf 3 einzige Conſtante iſt die Erhebung in den Abendſtunden mit Ausnahme der beiden vorletzten Tage).

13. Fall. Georg Kraus, 5 Jahre alt, wurde am 10. N 1873 mit vollſtändig entwickelter Meningitis in die Hei Kinderklinik aufgenommen.

| Fiebertabelle. | | |

Trübung. Die Theile ſind lebhaft vascularisirt und von zahlreichen größeren und kleineren, theils weißlichen, theils grauen Knötchen durchſetzt. Die beiden Seitenventrikel beträchtlich erweitert, namentlich in den hinteren Abſchnitten mit Serum gefüllt. Ependym verdickt, trübe, ſtark gekörnt. Außerdem noch Bronchitis, käſige Degeneration der Bronchialdrüſen und acuter Hydrocephalus.

Der Temperaturverlauf iſt in dieſem Falle ſo recht charakteriſtiſch für die Meningitis tuberculosa basilaris. Auch nicht eine Spur von Regelmäßigkeit findet ſich im Gange derſelben. Meiſt ſind es normale oder ſubnormale Temperaturen, nur am 11. früh, am 12. früh und am Todestage erreicht die Temperatur 39,6, 39,2 und 39,0. Ebenſo unregelmäßig und ohne Coordination an die Temperatur iſt die Pulsfrequenz.

14. Fall. Ludwig Häuſer, 4 Jahre alt, wurde am 25. April 1877 mit Caries der linken Tibia und chroniſcher Entzündung beider Tibio-Tarſalgelenke im Heidelberger Kinderſpital aufgenommen. Am 6. Juli zeigten ſich die erſten meningitiſchen Symptome.

Fiebertabelle.

	Mrgs.	Abds.			Mrgs.	Abds.	
Den 6. Juli	T. 38,6	39,2	Den 9. Juli	T. 39,0	38,8	P. 96	
» 7. »	» 38,8	38,6	» 10. »	» 38,2	39,0	» 80	108.
» 8. »	» 38,2	38,4 P. 88					

Exitus lethalis in der Nacht um 12 Uhr.

Sectionsbefund: Schädeldach dünn, aber compact. Dura mater haftet feſter. Die Pia iſt in ihrer ganzen Ausdehnung ſowohl an der Convexität als an der Baſis weißlich getrübt und ſtark ſerös infiltrirt, an der Baſis finden ſich auch in ihr einzelne miliare Knötchen. Die Venen der Pia ſind ſtrotzend gefüllt. Die Ventrikel enthalten mehr Serum wie gewöhnlich.

Anatomiſche Diagnoſe. Tuberkulöſe Baſilarmeningitis, Hyperämie des Gehirns, käſige Schwellung und Ulceration der Peyerſchen Drüſenhaufen, Schwellung der meſenterialen und Verkäſung der bronchialen Drüſen. Caries der rechten Tibia und chroniſche Entzündung beider Tibio-Tarſalgelenke.

Der Temperaturverlauf iſt ſehr unregelmäßig. Abendliche Exacerbationen wechſeln mit Remiſſionen ab. Morgens ſchwankt die Temperatur zwiſchen 38,2 und 39,0, Abends zwiſchen 38,4 und 39,2.

15. Fall. Johann Dicter, 4 Monate alt, wurde am 26. Oct. 1878 mit Catarrhus gastrointestinalis im Heidelberger Kinderhoſpital aufgenommen. Eintritt meningitiſcher Erſcheinungen am 18. Fbr. 1879.

Fiebertabelle.

	Mrgs.	Abds.
Den 18. Februar	T. 39,2	38,2
» 19. »	» 38,4	37,8
» 20. »	» 38,4	37,6

				Mrgs.	Abds.	
Den	21.	Februar	T.	38,6	37,8	P. 132
»	22.	»	»	37,8	37,8	
»	23.	»	»	37,4	37,8	
»	24.	»	»	38,4	—	
»	25.	»	»	38,4	38,2	P. 136
»	26.	»	»	38,4	38,6	» 132
»	27.	»	»	39,0	38,4	» 144
»	28.	»	»	38,6	38,4	» 120—136
»	1.	März	»	38,6	38,8	» 112
»	2.	»	»	38,4	39,2	» unzählbar
»	3.	»	»	38,8	39,2	» 170.

Am 4. März 7¼ Uhr Morgens trat der Tod ein, Temperatur 39,2, nach einer Viertelſtunde 39,2, von da ab alle 15 Minuten Meſſung 38,8—38,6—37,6—37,0.

Autopſie. Bei Eröffnung des Schädeldachs entleert ſich eine bedeutende Menge ſeröſer Flüſſigkeit. Die Venenſinus der Dura mater ſind mit dunkelrothem flüſſigem Blut gefüllt. Die Pia iſt in der ganzen Ausdehnung des Groß- und Kleinhirns ſerös infiltrirt. An der Gehirnbaſis und namentlich an den unterſten Abſchnitten der beiden Schläfenlappen nimmt dieſe Infiltration einen ſulzigen Charakter an. Die Pia erſcheint hier gelb-grünlich. Entlang den Gefäßen an der Hirnbaſis und auch an zahlreichen anderen Stellen finden ſich miliare graugelbe Knötchen. Die Seitenventrikel ſind ausgedehnt und mit Serum angefüllt, Ependym klar.

Anatomiſche Diagnoſe. Acute allgemeine Miliartuberkuloſe, acute tuberkulöſe Baſilarmeningitis, acuter interner Hydrocephalus, Circumſcripter Käſeherd mit centraler Erweichung im linken Unterlappen.

Temperatur ganz unregelmäßig, meiſt herrſchen abendliche Remiſſionen vor, nur vom 1. März ab iſt Abends die Temperatur höher als am Morgen, ſo auch am 26. Februar, wogegen am 22. Februar die Morgentemperatur der des Abends gleicht. Die Pulsfrequenz iſt der Temperatur nicht coordinirt, an den beiden letzten Tagen nimmt dieſelbe rapid zu.

16. Fall. Anton Hüngerle, 2½ Jahre alt, wurde am 24. Mai 1878 mit einer Fiſtel am linken Vorderarm im Heidelberger Kinderſpital aufgenommen, Eintritt meningitiſcher Erſcheinungen am 15. Dezember.

Fiebertabelle.

				Mrgs.	Abds.	
Den	16.	Dezember	T.	38,2	38,6	P. 144
»	17.	»	»	38,2	38,4	» 116
»	18.	»	»	38,0	38,4	» 128
»	19.	»	»	38,0	38,8	» 116

	Mrgs.	Abds.	
Den 20. Dezember T.	39,2	39,0	P. 142
» 21. » »	39,4	39,2	» 122
» 22. » »	38,8	38,4	» 110
» 23. » »	39,2	—	» 144.

Exitus lethalis am 24. Morgens 5½ Uhr.

Section. Schädel groß. Große Fontanelle in geringerem Grade, kleine Fontanelle weit offen. Dura mater in der Mittellinie adhärent. Pia mater ſchwach trüb, an mehreren Stellen mit kleinen grauen Knötchen beſetzt. An der Hirnbaſis iſt dieſe Trübung ſtärker ausgeſprochen und zeigen ſich hier die Maſchen der weichen Hirnhäute hochgradig ödematös. Die Trübung geht in eine eitrige Infiltration über an der Oberfläche des Kleinhirns an der großen Hirnſpalte. Hirnventrikel ſehr ſtark erweitert und mit klarer Flüſſigkeit gefüllt.

Anatomiſche Diagnoſe. Meningitis tuberculosa, Conglomerattuberkel in der Großhirnrinde und des rechten Seh- und Streifenhügels, Hydrocephalus internus, Conglomeratknoten der rechten Lunge, Obliteratio Pericardii, fibröſe Pleuritis rechts mit bindegewebiger Adhäſion des rechten vorderen Lungenrandes, käſige Oſtitis und Perioſtitis des rechten Ringfingers und Perioſtitis der linken Ulna.

Temperatur ſchwankend, am 22. iſt die Temperatur niedriger als an den zwei vorhergehenden Tagen. Meiſt conſtante Abendexacerbationen (Ausnahmen 20., 21. u. 22.), Pulsfrequenz ohne Beiordnung an die Temperatur.

17. Fall. Amadeus Keller, 2 Jahre 4 Monate alt, wurde am 18. Auguſt 1880 mit Rhachitis und chroniſch-käſiger Pneumonie im Heidelberger Kinderſpital aufgenommen. Eintritt meningitiſcher Erſcheinungen gegen den 14. Dezember.

Fiebertabelle.

	Mrgs.	Abds.	
Den 14. Dezember T.	38,6	38,0	P. 120
» 15. » »	37,8	38,3	» 96—108
» 16. » »	37,1	38,4	» 96
» 17. » »	37,4	38,4	» 76
» 18. » »	37,8	38,5	» 80—100

	Mrgs.	Mttgs.	Abds.		Mrgs.	Mttgs.	Abds.
Den 19. Dezember T.	37,6	38,8	38,6	P.	87	90	90
» 20. » »	38,3	38,4	38,8	»	80	118	120
» 21. » »	39,2	40,2	39,4	»	jagend	—	116
» 22. » »	um	6 Uhr	Mrgs.		39,8		
	»	8 »	»		39,8		
	»	12 »	Mttgs.		39,6	P. 165	
	»	2 »	»		39,6		
	»	4 »	»		39,8		

Den 22. Dezember T.	um	6	Uhr	Abds.	39,4 P. 162
»	»	8	»	»	39,0
»	»	10	»	Nachts	39,2
»	»	½12	»	»	39,6.

Exitus lethalis um ½12 U. Nachts. Die poſtmortale Meſſung ergab 10 Min. nach dem Todeseintritt 39,5 und weiter immer Abfall.

Section am 23. Dezember.

Pia beider Großhirnhemiſphären ſerös infiltrirt. An der Gehirnbaſis iſt die Pia eitrig infiltrirt und mit zahlreichen gelbgrauen Knötchen durchſetzt, dieſelbe Veränderung iſt vom Chiasma NN. opt. längs der Brücke bis zur Medulla oblongata und in den Sylviſchen Gruben. Die beiden Seitenventrikel ſind beträchtlich erweitert und enthalten eine große Menge trüber Flüſſigkeit.

Der Temperaturverlauf in dieſem Falle ſehr ſchwankend, nur herrſchen (mit Ausnahme des 14. und des Todestages) conſtante Abendexacerbationen vor. Die Temperatur ſchwankt Morgens zwiſchen 37,1 und 39,8, Mittags und Abends zwiſchen 38,0 und 40,2. Den Höhepunkt erreicht die Temperatur am Mittag des vorletzten Tages (40,2), wogegen ſie am Todestage nur bis 39,8 ſich erhebt. Die Pulsfrequenz iſt zwar nicht ganz unregelmäßig, aber ohne Coordination an die Temperatur.

18. Fall. Caesarine Breitenſtein wurde am 6. Juni 1882 mit Bronchitis capillaris und entwickelter Meningitis im Heidelberger Kinderſpital aufgenommen; das Kind iſt 4 Jahre alt.

Fiebertabelle.

			Mrgs.	Abds.	
Den	6.	Juni T.	—	39,5	P. 124
»	7.	» »	39,6	39,7	» 140
»	8.	» »	38,9	38,5	» —
»	9.	» »	39,0	39,0	» —
»	10.	» »	38,8	39,2	» 136
»	11.	» »	39,0	39,4	» 132
»	12.	» »	39,5	39,7	» 180.

Exitus lethalis ½12 Uhr Nachts. Section am 13. Juni Nachmittags 5 Uhr.

Dura mater am Schädel etwas feſter, im Sinus longitud. flüſſiges Blut. Pia blutreich, ſtark ſerös infiltrirt. Rechts zeigt die piale Auskleidung der Großhirnhemiſphären eine theils mehr gleichmäßige eitrige Infiltration, theils Durchſetzung mit kleinen käſigen Knötchen, links dieſelbe Veränderung etwas weiter nach hinten und in geringerem Grade. Piale Auskleidung der Fossae Sylvii gleichfalls ſehr blutreich und Durchſetzung mit kleinen Knötchen. Die Seitenventrikel ſind ſtark erweitert und mit Flüſſigkeit angefüllt.

Anatomiſche Diagnoſe. Tuberkulöſe Meningitis, acuter Hydro-

cephalus internus, käſige Degeneration der Lymphdrüſen, diſſeminirte Miliartuberkuloſe der Lunge, Milz, Leber, Nieren.

Der Temperaturverlauf iſt ſehr ſchwankend, am 8. iſt die Temperatur niedriger als an den vorhergehenden Tagen, ſo auch am 10. Morgens niedriger als am 9. Morgens. Mit Ausnahme des 8. und 9. ſind conſtante Abendexacerbationen. Die Pulsfrequenz iſt der Temperatur nicht ganz coordinirt.

19. Fall. Martin Weigel, 5 Jahre alt, wurde am 6. Novbr. 1877 mit Coxitis dextra in die Heidelberger Kinderklinik aufgenommen. Eintritt meningitiſcher Erſcheinungen gegen den 16. Dezember.

Fiebertabelle.

		Mrgs.	Abds.			
Den 16.	Dezember	T. 38,6	39,6	P.	—	—
» 17.	»	» 39,0	40,0	»	104	—
» 18.	»	» 39,4	40,2	»	—	—
» 19.	»	» 39,0	38,8	»	—	—
» 20.	»	» 39,0	40,2	»	—	—
» 21.	»	» 38,4	39,0	»	100	—
» 22.	»	» 38,0	38,4	»	80	—
» 23.	»	» 37,0	38,6	»	76	80
» 24.	»	» 37,4	38,8	»	60	68
» 25.	»	» 37,2	38,0	»	—	—
» 26.	»	» 37,0	39,2	»	76	—
» 27.	»	» 39,6	40,4	»	100	—
» 28.	»	» 39,4	39,6	»	100	—
» 29.	»	» 39,0	40,2	»	—	—
» 30.	»	» 40,0	39,6	»	144	—
» 31.	»	» 39,2	38,8	»	140	—
» 1.	Januar	» 37,6	39,2	»	180	—
» 2.	»	» 38,0	40,0	»	140	—

Um 6 Uhr trat der Tod ein.

Die poſtmortale Meſſung ergab 10 Minuten nach dem Tode 40,0, je 10 Minuten weiter 39,4—39,0—38,8—38,4—38,0—37,8—37,2—37,2—36,6.

Section am 3. Januar.

Die Venen der Pia ſtark mit Blut gefüllt. Die Pia entſprechend der Convexität des Gehirns klar und durchſcheinend, an der Baſis jedoch beſonders über dem Pons bis zum Chiasma NN. opt. getrübt und verdickt und mit miliaren Einſprengungen durchſetzt. Beſonders zahlreich ſind letztere in der Fossa Sylvii, wo ſie ſich deutlich an dem Verlauf der Gefäße halten. Die Seitenventrikel ſind ſtark erweitert und mit ſchwachtrübem Serum gefüllt. In der Subſtanz des Kleinhirns finden ſich in beiden Hemiſphären käſige Knoten bis zur Größe einer Erbſe eingebettet.

Anatomiſche Diagnoſe. Meningitis tuberculosa basilaris, Tuber-

[illegible] Lungen, Nieren und Gehirns, Atelektaſe der Lungen, [illegible]leuritis rechts, Gehirnödem, Oſteomyelitis i. d. Epiphyſe [illegible]ur.

[illegible] peraturverlauf äußerſt unregelmäßig. Bis zum 21. De[illegible] das Fieber ſehr hoch, von da ab bis zum 27. iſt die [illegible]tur Morgens normal, Abends nur mäßig erhöht, vom 27. [illegible] dieſelbe wieder hohe Grade bis zum Lebensende. Die [illegible] Grade fallen auf den 17., 18., 20., 27., 29., 30. und auf [illegible]stag, an welchem letzten Tage die Temperatur am Morgen [illegible] nur 38,0 aufweiſt. Die Exacerbationen fallen faſt immer [illegible]men 19., 30. und 31.) auf den Abend. Die Pulsfrequenz [illegible] jedwede Spur von Coordination an die Temperatur, be[illegible]swerth iſt die Abnahme derſelben am Todestage bei einer [illegible]aturhöhe von 40,0 auf 140 Schläge, während ſie Tags vorher [illegible] Temperaturhöhe von 39,2 180 Schläge aufwies.

[illegible] Fall. Friedrich Nichtern, 2 Jahre alt, wurde am 15. April [illegible] chroniſcher Pneumonie, chroniſcher Otitis und bereits [illegible]elter Meningitis tuberculosa basilaris in die Heidelberger [illegible]linik aufgenommen.

[illegible]tabelle.	Mrgs.	Abds.		Mrgs.	Abds.
[illegible] April T.	38,4	39,0	Den 10. April T.	38,6	38,6
[illegible]	38,3	39,4	» 11. » »	38,4	38,8
[illegible]	38,6	38,8	12. » »	38,0	38,0 P. 88
[illegible]	38,4	38,8	» 13. » »	37,1	38,2 » 92.

[illegible] Mrgs.	38,0		16. April T. Mrgs.	3, 39,0	
Mttgs.	12, 38,2	P. 144	» » » »	6, 38,5	
[illegible]	3, 38,4		» » » Mttgs.	12, 38,2	P. 152—160
Abds.	6, 38,4		» » » »	3, 38,3	
	9, 38,2		» » » Abds.	6, 38,4	
	12, 39,5		» » » »	9, 38,4	
Mrgs.	3, 39,6		» » » »	12, 39,2	
	6, 39,8		17. » » Mrgs.	3, 39,8	
Mttgs.	12, 38,6	P. 164	» » » »	6, 40,0	
[illegible]	3, 38,7		» » »	9, 40,0	
Abds.	6, 38,8		» » » Mttgs.	12, 40,2	
[illegible]	9, 38,5		» » » »	3, 39,4	

12, 39,4 gemeſſen während des Sterbens Abds. $5^3/_4$, 40,0 P. jagend, unzählbar.

[illegible] Minuten post mortem 39,4, 20 Minuten post mortem 39,2. [illegible] am 18. April Morgens 10 Uhr.

[illegible]ater feſter als gewöhnlich am Schädel haftend, auf der [illegible] ſitzt an der Dura, von dieſer ausgehend, eine circa [illegible] Platte, am Großhirn dementſprechend eine Ver[illegible] Pia mit der Oberfläche des Tumors verwachſen. Die

Pia und Arachnoides an der Schädelbaſis ſind, insbeſondere aber entſprechend dem Pons, Chiasma NN. opt. und Sylviſchen Gruben, eitrig infiltrirt und mit miliaren Knötchen durchſetzt. Die Seitenventrikel ſind beträchtlich erweitert und enthalten trübe Flüſſigkeit.

Anatomiſche Diagnoſe. Tuberkulöſe Baſilarmeningitis, Conglomeratknoten der Dura mater, ausgebreitete käſige Degeneration der Lymphdrüſen, Tuberkel der Lunge, Leber, Nieren, Milz und der ſeröſen Häute, Otitis, catarrhaliſche Pneumonie beider Unterlappen und Höhlenbildung in der rechten Lungenſpitze.

Temperaturverlauf ſehr unregelmäßig, an den beiden erſten Krankheitstagen iſt dieſelbe höher als an den folgenden bis zum 14. Nachts. Abends iſt die Temperatur indeſſen conſtant etwas erhöht, nur am 10. und 12. iſt die Abendtemperatur der des Morgens gleich. Am Todestage hält ſie ſich bis zum Todeseintritt auf 40, nur in der dritten Morgen- und Mittagſtunde fällt ſie in etwas. Die Pulsfrequenz iſt ohne Coordination an die Temperatur.

21. Fall. Ein Knabe von 5 Jahren wird mit chroniſcher Entzündung und Vereiterung des rechten Kniegelenks ins Spital aufgenommen.

Fiebertabelle.

	Mrgs.	Abds.	Mrgs.	Abds.		Mrgs.	Abds.	Mrgs.	Abds.
1. Tag	T. 38,0	39,0	P. 88	94	6. Tag.	T. 38,4	38,5	P. 100	132
2. »	» 38,2	38,6	» 70	74	7. »	» 37,8	39,2	» 84	158
3. »	» 38,4	38,4	» 82	80	8. »	» 38,4	38,0	» 134	100
4. »	» 38,0	39,0	» 84	96	9. »	» 38,2	—	» 116	—
5. »	» 38,5	38,4	» 80	82	10. »	» 38,8	38,8	» 120	110.

Am 11. Tage früh erfolgte der Exitus lethalis unter einfachen Symptomen von Collaps. T. 40,0, P. unzählbar.

Die Meſſung betrug eine Viertelſtunde post mortem 40,4, eine Stunde nach demſelben 39,4.

Section 26 Stunden nach dem Tode.

Dura nicht adhärent, Sinus ſtark gefüllt. Grüngelbes reichliches Exſudat in den Maſchen der Pia vom Chiasma bis an den Pons. Pia der Brücke ſtark getrübt. Piablätter der Fossae Sylvii auf beiden Seiten verlöthet. Sehr beträchtlicher, trüber, ſeröſer Erguß in den erweiterten Seitenventrikeln. Sonſt noch in den Lungen Emphyſem, Bronchitis catarrhalis, beginnende Bronchopneumonie und ein erweichter Herd. Im rechten Kniegelenk Eiterung, Zerſtörung der Knorpel und Caries der Gelenkflächen.

Temperaturverlauf unregelmäßig, am 1. Tage iſt Abends die Temperatur höher als an den folgenden 2 Tagen, dann wiederholt

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[21]) Steffen, Handbuch der Kinderkrankheiten. Bd. 5. II. pag. 444. Tübingen 1880.

ſich wieder dieſes Verhalten der Temperatur. Bald fallen die Exacerbationen auf den Morgen bald auf den Abend, dann ſind wieder Morgen- und Abendtemperaturen gleich. Vor dem Tode ſteigt die Temperatur plötzlich auf 40,0, welche Höhe ſie während des ganzen Verlaufs nicht erreichte. Die Pulsfrequenz iſt ohne jedwede Coordination an die Temperatur.

22. Fall. Ein Mädchen von 4 Jahren wird am 19. Juni 1867 wegen Conjunctivitis, Keratitis und Otitis interna chronica im Spital aufgenommen. Gegen Ende Auguſt ſtellten ſich die Prodromalerſcheinungen der tuberkulöſen Meningitis ein.

Fiebertabelle.

	Mrgs.	Abds.	Mrgs.	Abds.		Mrgs.	Abds.	Mrg.	Abds.
30. Aug.	T. 38,0	38,5	P. 100	100	5. Sept.	T. 38,0	38,5	P. 100	102
31. »	» 38,0	35,5	» 100	112	6. »	» 37,0	38,0	» 88	110
1. Sept.	» 39,0	38,4	» 100	100	7. »	» 37,5	38,0	» 100	80
2. »	» 38,0	38,0	» 100	106	8. »	» 38,0	37,3	» 68	90
3. »	» 38,6	38,5	» 104	104	9. »	» 37,0	39,5	» 76	80
4. »	» 39,0	38,5	» 106	112	10. »	» 41,0	—	» 52	—.

Am Nachmittag trat der Tod ein. Eine Stunde post mortem betrug die Temperatur 37,0.

Section 20 Stunden nach dem Tode.

Dura mäßig adhärent. Sinus mit dünnflüſſigem Blut gefüllt. Dura über dem Os petros. dextr., dem Clivus und der Sella turcica etwas verdickt und mißfarbig. Caries des rechten Os petrosum. Auf der ganzen Gehirnbaſis befindet ſich in der Pia ein eitriges Exſudat in großer Menge, dieſes Exſudat erſtreckt ſich auch hinein in die Fossae Sylvii. Im Exſudat eingebettet eine Unmaſſe miliarer Tuberkel, ebenſolche Veränderung an der baſalen Pia des Cerebellum und Pia der unteren Seite der Medulla oblongata. Die Seitenventrikel erweitert und mit trübem, ſerös-eitrigem Erguß gefüllt. Sonſt noch Tuberkel in beiden Lungen und Verkäſung der Bronchial- und Mediaſtinaldrüſen.

Im Temperaturverlauf iſt auch nicht eine Spur von Regelmäßigkeit. Während des ganzen Verlaufs ſteigt ſie nur am 1. September Morgens und am 9. September Abends auf 39,0 und 39,5. Am Todestage erhebt ſie ſich plötzlich bis auf 41,0, während, was ſehr auffallend iſt, die Pulsfrequenz auf 52 ſinkt; auch ſonſt iſt die Frequenz des Pulſes der Temperatur in keiner Weiſe coordinirt.

23. Fall. Ein Knabe von 3 Jahren wird am 2. Februar 1874 wegen Maſtdarmvorfall ins Kinderſpital aufgenommen. Allmälig entwickelt ſich unter fiebrigen Erſcheinungen, welche Ende Mai

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[22]) Handbuch der Kinderkrankheiten. Bd. 5. II. pag. 455. [23]) Handbuch der Kinderkrankheiten. Bd. 5. II. pag. 462.

wieder ceſſiren, chroniſche Pneumonie in beiden Lungen. Am 10. Juli tritt plötzlich wieder Fieber auf, Kopfſchmerzen, Erbrechen.

Fiebertabelle.	Mrgs.	Mttgs.	Abds.		Mrgs.	Mttgs.	Abds.
Den 10. Juli T.	—	—	39,8	P.	—	—	128
» 11. » »	38,9	39,4	39,9	»	100	116	116
» 12. » »	38,5	39,6	38,7	»	108	100	88
» 13. » »	38,5	38,6	38,1	»	68	68	56
» 14. » »	37,6	38,2	38,4	»	58	60	60
» 15. » »	37,9	38,3	38,1	»	60	72	88
» 16. » »	37,7	39,1	39,4	»	84	96	100
» 17. » »	39,5	39,0	39,4	»	112	128	124
» 18. » »	39,4	39,3	39,2	»	124	148	152
» 19. » »	38,8	38,7	39,9	»	156	164	168
» 20. » »	38,0	39,9	39,0	»	164	164	168.

Der Tod erfolgte Nachts 12 Uhr ohne voraufgegangenen Krampfanfall.

Die poſtmortale Meſſung betrug eine Viertelſtunde nach dem Tode 41,9, 15 Minuten ſpäter 40,4.

Section am 22. Juli Vormittags.

Dura dem Schädeldach feſt adhärent, namentlich in der Gegend des linken Tuber parietale, Sinus leer. Sowohl an der Convexität als an der Baſis ſulzig-eitriges Exſudat in mäßiger Menge, welches hauptſächlich in den Maſchen der Pia angeſammelt iſt. Reichlicher Erguß in beiden Seitenventrikeln. Außerdem chroniſche Pneumonie beider Lungen und friſchere käſige Peribronchitis; ſowie Tuberkuloſe der meiſten Organe.

Temperaturverlauf ohne Spur von Regelmäßigkeit. Manchmal fallen die höheren Temperaturen auf den Abend; dann wieder auf den Mittag oder Morgen, und auch ſonſt iſt im Verlaufe der Temperatur die größte Unregelmäßigkeit. Post mortem erhebt ſich die Temperatur, die praemortal 39,9 nicht überſchreitet, auf 41,9, alſo um 2 volle Grade. Die Pulsfrequenz iſt der Temperatur in keiner Weiſe coordinirt.

24. Fall. Otto A., $2^1/_4$ Jahre alt, wurde am 24. November 1876 aufgenommen. Patient hat bereits Maſern und Scharlach überſtanden und vor einem Jahre einmal an Krämpfen gelitten.

Am 24. T. 36,8, P. 75; in den nächſten Tagen war die Temperatur Morgens ziemlich normal, Abends ſtieg ſie bis auf 38,4, die Pulsfrequenz ſchwankte zwiſchen 80 und 160 und war mehr oder minder unregelmäßig. Am Abend des 29. ſtieg die Temperatur auf 39,1, am folgenden Tage auf 40,5, während der Puls unzählbar ſchnell und klein wurde. Nachmittags 6 Uhr erfolgte der Tod.

[24]) Henoch, Charité-Annalen. N. F. 4. pag. 489.

Section. Multiple Adhäſionen zwiſchen Dura und Pia. Die Pia der Baſis ſtark durchfeuchtet, mit einer trüben grauweißen Maſſe ſtark infiltrirt und von zahlreichen miliaren Knötchen durchſetzt. Die Seitenventrikel mit farbloſer Flüſſigkeit gefüllt. Tuberkuloſe des Gehirns.

25. Fall. Max D., 2 Jahre alt, aufgenommen am 17. April 1874 mit einem großen Abſceß auf der Epiphyſe des rechten Unterarms und einem zweiten am unteren Ende des rechten Unterſchenkels. Vom 2. Mai an Fieber.

T. am 2. Mai 39,1, P. 152; am 3. Mai T. 40,0, P. 170; am 4. Mai T. 42,0; Tod in der Nacht.

Section. Im Groß- und Kleinhirn Tuberkel. In der Pia der Baſis gallertige Infiltration und miliare Knötchen. Ventrikel weit mit Serum angefüllt. Bronchopneumonie beider Unterlappen und theilweiſe Verkäſung der Bronchialdrüſen.

26. Fall. Martha Sp., 1 Jahr alt, ſoll wiederholt an Convulſionen gelitten haben, aufgenommen den 11. Juli 1877.

Am 11. Juli T. 38,6, P. 160; am 12. Juli T. 39,7, P. 172. Tod in der Nacht.

Section. Zahlreiche miliare Knötchen in der Pia der Baſis, wie auch der Convexität. Ventrikel mit Serum angefüllt und ſtark erweitert. Tuberkuloſe des Gehirns und der verſchiedenſten Organe, ſowie alte adhäſive Pleuritis rechts.

27. Fall. Auguſte W., 5 Jahre alt, aufgenommen am 2. Jan. mit den Symptomen einer vorgeſchrittenen tuberkulöſen Meningitis.

Fiebertabelle.

	Mrgs.	Abds.	
Den 3. Januar	T. 38,2	36,7	P. 144
» 4. »	» 38,7	37,1	» —
» 5. »	» 38,5	39,4	» 184—192—200
» 6. »	» 38,5	39,4.	

Am 6. Abends trat der Tod ein.

Section. Ausgedehnte Meningitis tuberculosa mit zahlreichen miliaren Knötchen an der Baſis; auf der Convexität beider Frontallappen, beſonders längs der größeren Gefäße viele graue Knötchen, aber ohne Exſudat in der Pia. Hydrocephalus aller Ventrikel mit Erweichung der umgebenden Markſubſtanz. Im Pons Varoli ein erbſengroßer Tuberkel. Tuberkuloſe der meiſten übrigen Organe und Vulvitis gangraenosa.

28. Fall. Alfred J., 1 Jahr alt, aufgenommen am 11. April 1876, früher geſund, ſoll nach Ausſage der Mutter ſeit einigen Tagen apathiſch ſein und «Krämpfe» haben.

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[25]) Charité-Annalen. Bd. IV, pag. 498. [26]) Ebendaſelbſt. pag. 494. [27]) Ebendaſelbſt. pag. 499. [28]) Ebendaſelbſt. pag. 501.

Am 11. April T. 36,5, P. 72, in den nächſten 2 Tagen keine Temperaturerhöhung, P. 88—120. Erſt am 14., 15. und 16. ſteigt die Temperatur, die Morgens normal iſt, Abends auf 38,8, am 17. ſogar auf 39,5 mit entſprechender Zunahme der Pulsfrequenz (136—180 in den Abendſtunden, Morgens 80—132). Den 18. T. 40,8, P. über 200, leichte allgemeine Zuckungen. Tod.

Section. Meningitis tuberculosa, an der Baſis und Convexität mit zahlreichen miliaren Knötchen Pia durchſetzt, auch in den Plexus chorioidei der Ventrikel. Im Hinterlappen der linken Hemiſphäre ein taubeneigroßer tuberkulöſer Herd in der Rindenſchichte. Hydrocephalus externus et internus. Miliartuberkuloſe der meiſten Organe.

29. Fall. M. Henriette, 8 Monate alt, wurde am 21. Febr. 1877 auf die Klinik gebracht, ſoll zuweilen an Convulſionen gelitten haben. T. etwas erhöht, P. unregelmäßig. Trouſſeau'ſche Flecken, am 23. Februar T. 41,2, P. ſehr klein und ſchnell, nach wenigen Stunden Tod.

Section. Tuberkulöſe Baſilarmeningitis, Extravaſate zwiſchen den Gyri des Mittel- und Stirnlappens. Tuberkel auf dem Ependym der Seitenventrikel, chroniſcher Hydrocephalus mit darauf gefolgtem acutem. Außerdem Miliartuberkuloſe der verſchiedenſten Organe und Magenerweichung.

30. Fall. Ein 4jähriges Kind, Namens Lumpe, wird am 19. April 1876 in die Kinderklinik aufgenommen.

Fiebertabelle.

	Mrgs.	Abds.		
Den 22. April T.	—	38,5	P.	96
» 23. » »	37,8	38,9	»	68
» 24. » »	37,5	37,8	»	64— 80
» 25. » »	38,4	38,7	»	88— 96
» 26. » »	38,2	38,7	»	96—108
» 27. » »	38,0	38,4	»	—
» 28. » »	38,2	38,0	»	—
» 29. » »	38,5	39,0	»	112
» 30. » »	40,2	40,4	»	180.

Dauer der Krankheit 12 Tage. Temperatur ohne geſetzmäßigen Verlauf, am 24., 27. und 28. niedriger als zuvor, die höchſte Ziffer (38,9) ſchon am 5. Krankheitstage erreichend. Am vorletzten Abend plötzliche Steigerung auf 39,0, am Todestage auf 40,2 und 40,4 ſteigend. Sonſt immer zwiſchen 37,5 und 38,7 ſchwankend, mit geringen abendlichen Erhebungen, mit Ausnahme des 28., wo die Abendtemperatur etwas niedriger iſt. Die Pulsfrequenz iſt nicht durchweg der Temperatur coordinirt.

[29]) Handb. d. Kinderkrankh. Bd. 4. II. p. 437. [30]) Charité-Annalen. N. F. IV. p. 507.

31. Fall. Ein 3 Jahre altes Kind, Namens Dahn, aufgenommen am 4. Februar 1878 mit Pleuritis chronica sinistra.

Fiebertabelle.

		Mrgs.	Abds.		Mrgs.	Abds.
Den	5. Februar	T. 38,0	39,0	P.	108	132
»	6. »	» 37,8	38,9			
»	7. »	» 38,2	39,3			
»	8. »	» 37,9	38,9	»	120	144
»	9. »	» 38,0	38,3			
»	10. »	» 38,0	38,3	»	108	—
»	11. »	» 37,5	38,1	»	96	108
»	12. »	» 37,0	38,0	»	—	—
»	13. »	» 38,0	40,2	41,0.		

Krankheitsdauer etwa 10 Tage. Temperatur ganz unregelmäßig, in den Morgenſtunden entweder normal oder mäßig erhöht, Abends immer höher, bis höchſtens 39,3. Am letzten Nachmittag plötzliche Steigerung bis 41,0. Die Pulsfrequenz iſt ohne Beiordnung an die Temperatur.

32. Fall. Handwein, 4 Jahre alt, aufgenommen am 6. April 1878 mit allgemeiner Tuberkuloſe, zumal des Bauchfells, fühlbaren Schwellungen der Meſenterialdrüſen, Apathie, Somnolenz etc.

Fiebertabelle.

			Mrgs.	Abds.		
Den	6. April	T.	—	38,5	P. 96	
»	7. »	»	37,5	38,0	» 96	
»	8. »	»	37,2	36,8	» 72	88
»	9. »	»	38,4	38,5	» 80	104
»	10. »	»	36,8	37,5	» 64	
»	11. »	»	38,0	38,1	» 120	132
»	12. »	»	38,2	38,6	» 128	136
»	13. »	»	38,5			
»	» »	11 Uhr »	39,2			
»	» »	4 »	39,8	P. 175.		
»	» »	6 »	40,3			
»	» »	9 »	41,8			

Krankheitsdauer etwa 8 Tage. Temperatur ſehr ſchwankend, erſt am Nachmittage des Todestages 39 erreichend und nun ſtündlich bis 41,8 ſteigend. Die Pulsfrequenz iſt auch hier meiſt nicht im Verhältniß zur Temperatur.

33. Fall. Ein $1^3/_4$ Jahre altes Kind wird im zweiten Stadium (tiefe Somnolenz etc.) am 16. Dezember 1877 aufgenommen.

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[31]) Charité-Annalen. Bd. IV. pag. 507. [32]) Ebendaſelbſt. pag. 508. [33]) Ebendaſelbſt. pag. 508.

Fiebertabelle.			Mrgs.	Abds.		
Den 11.	Dezember	T.	37,3	37,3	P. 72	
» 12.	»	»	37,5	38,2	» 78	124
» 13.	»	»	38,5	38,5	» 116	
» 14.	»	»	38,1	38,8	» 146	160
» 15.	»	»	38,2	38,9	» 144	
» 16.	»	»	38,0	39,5	» 190	
» 17.	»	»	40,0	—	» 210.	

Auch in dieſem Falle finden wir einen ſchwankenden Temperaturverlauf. Meiſt ſind abendliche Exacerbationen, bisweilen (den 11. und 13.) ſind die Abendtemperaturen denen des Morgens gleich. Am Todestage Steigerung bis auf 40,0 mit einer Pulsfrequenz von 210, ſonſt iſt auch in dieſem Falle keine Coordination des Pulſes an die Temperatur vorhanden.

34. Fall. Ein 4jähriges Kind, Namens Hänel, erkrankte vor 8 Tagen, aufgenommen am 6. Juni 1876.

Fiebertabelle.			Mrgs.	Abds.		
Den 6.	Juni	T.	—	38,2	P. 76	
» 7.	»	»	38,8	39,0	» 112	
» 8.	»	»	38,7	38,9	» 112	
» 9.	»	»	37,5	39,4	» 124	132
» 10.	»	»	37,4	38,5	» 124	132
» 11.	»	»	38,2	38,0	» 164	
» 12.	»	»	38,5	40,0	» 208.	

Temperaturverlauf ganz unregelmäßig, am 9. und 10. erheblich niedriger als zuvor, abendliche Exacerbationen (mit Ausnahme des 11.), am Todestage rapide Steigung auf 40,0 mit einer Pulsfrequenz von 208. Sonſt iſt die Pulsfrequenz in keinem Verhältniß zur Temperatur.

35. Fall. Ein $^1/_2$jähriges Kind, aufgenommen am 7. März 1878. Dauer der Krankheit etwa 14 Tage.

Fiebertabelle.			Mrgs.	Abds.	
Den 7.	März	T.	38,0	38,3	P. 128
» 8.	»	»	38,0	37,5	» 160
» 9.	»	»	37,3	38,6	
» 9.	»	»	—	39,3	» 212
» 9.	» $12^1/_2$ Nachts	»	—	40,6.	

Temperaturverlauf unregelmäßig, am 9. iſt die Temperatur niedriger als an den vorhergehenden Tagen, am Todestage Nachts plötzlich auf 40,6 ſteigend. Pulsfrequenz nicht der Temperatur beigeordnet.

[34]) Charité-Annalen. Bd. IV. pag. 508. [35]) Ebendaſelbſt. pag. 509.

36. Fall. C., $8^1/_2$ Jahre alt, eingetreten den 30. April 1875 am 8. Krankheitstage.

Fiebertabelle.	Mrgs.	Abds.	
Den 30. April T.	—	39,0	P. 110—120
» 1. Mai »	37,6	39,0	» 80— 60—90
» 2. » »	38,0	38,0	» 96—140
» 3. » »	38,4	38,6 39,0	» 150—150

Todeseintritt am 4. Mai. Section am 5. Mai, 25 Stunden nach dem Tode.

Pia mater ist infiltrirt mit einer serösen trübweißen Flüssigkeit. Im Gehirn finden sich Tuberkel, an der Gehirnbasis mikroskopisch keine Granulationen nachweisbar. Die Seitenventrikel enthalten eine kleine Quantität Serum.

Temperaturverlauf unregelmäßig, am 2. und 3. ist Abends die Temperatur niedriger als an den vorhergehenden Abenden. Die Pulsfrequenz ist der Temperatur nicht coordinirt.

37. Fall. R., 7 Jahre alt, eingetreten ins Hospital am 16. September 1875, schon seit 8 Tagen krank.

Fiebertabelle.	Mrgs.	Abds.		Mrgs.	Abds.
Den 16. September T.	38,0	39,0	P.	95	92
» 17. » »	37,6	38,0	»	68	84
» 18. » »	37,0	38,0	»	74	78
» 19. » »	37,0	37,6	»	60	72
» 20. » »	37,0	38,0	»	60	65
» 21. » »	37,6	39,0	»	98	158
» 22. » »	38,0	38,4	»	144	158—180
» 23. » »	38,2	39,0	»	180	172—164
» 24. » »	38,5	39,7	»	180	172.

Todeseintritt um 1 Uhr in der Nacht.

Temperaturverlauf schwankend, am 16. und 21. ist die Temperatur höher als in den darauffolgenden Tagen (bis zum 23.). Vom 23. ab erreicht sie Abends hohe Grade, die bis zum Tode andauern. Das einzige Constante sind die Abendexacerbationen. Die Pulsfrequenz steht meist in keinem Verhältniß zur Temperatur.

38. Fall. Th. Pauline, 4 Jahre alt, seit 15 Tagen krank, eingetreten den 22. September.

Fiebertabelle.	Mrgs.	Abds.		Mrgs.	Abds.
Den 22. September T.	—	39,2	P.	—	112
» 23. » »	37,6	39,0	»	170	158
» 24. » »	39,0	39,2	»	156	166

[36]) Boutan, Op. cit. p. 13. [37]) Ebendas. p. 17. [38]) Ebendas. p. 19.

		Mrgs.	Abds.		Mrgs.	Abds.
Den 25.	September T.	38,6	39,4	P.	166	166
» 26.	» »	39,0	39,8	»	158	160
» 27.	» »	39,4	40,4	»	140	180
» 28.	» »	39,0	40,0	»	178	212
» 29.	» »	38,8	39,2	»	210—190—186—183—160	
» 30.	» »	39,2	40,0	»	176	200.

Temperaturverlauf ganz unregelmäßig, am 25. Morgens ift die Temperatur niedriger als am vorhergehenden Tage, ebenfo find die Temperaturen vom 27. und 28. bei weitem höher als am folgenden Tage. Die höchften Ziffern erreicht die Temperatur am 27., 28. und am Todestage Nachmittags. Die Pulsfrequenz nicht coordinirt, fo z. B. ift die Frequenz des Pulfes am 29. bei einer T. von 38,8 210, während fie am Abend bei einer T. von 39,2 nur 160 ift.

39. Fall. M., 9 Jahre alt, aufgenommen den 16. September mit allgemeiner Tuberkulofe und entwickelter Meningitis.

		Mrgs.	Abds.		Mrgs.	Abds.
Den 16.	September T.	39,8	39,4	P.	120	96
» 17.	» »	39,0	39,4	»	159	156
» 18.	» »	40,0	40,0	»	180	210
» 19.	» »	40,0	41,0	»	180	180
» 20.	» »	40,2	41,6	»	180	140—120—100.

Die poftmortale Meffung betrug in der Achfelhöhle 41,7, in der Vagina 42,6.

Temperaturverlauf ungefetzmäßig, am Morgen des 17. ift die Temperatur niedriger als am Morgen des 16., am 18. Morgen- und Abendtemperatur gleich, von welchem Tage ab fie regelmäßiger wird und nicht unter 40 finkt. Die Pulsfrequenz ift nicht an die Temperatur coordinirt, fo z. B. zeigt diefelbe am Todestage Morgens bei einer Temperatur von 40,0 180 Schläge in der Minute, während fie am Abend bei einer Temperatur von 41,6 bis auf 100 Schläge finkt.

40. Fall. Ein 9jähriges Kind, Anfang der Krankheit am 27. Mai, eingetreten ins Hofpital am felben Tage.

Fiebertabelle.

		Mrgs.	Abds.		Mrgs.	Abds.
Den 27.	Mai T.	—	37,0	P.	—	68
» 28.	» »	38,0	38,4	»	90	95
» 29.	» »	38,2	38,1	»	94	96
» 30.	» »	38,5	38,7	»	90	94
» 31.	» »	38,5	39,0	»	92	96

[39]) Boutan, op. cit. pag. 28. [40]) Archambault, op. cit. pag. 597.

	Mrgs.	Abds.		Mrgs.	Abds.
Den 1. Juni	T. 38,7	38,9		P. 86	86
» 2. »	» 38,5	39,2		» 90	98
» 3. »	» 38,7	39,4		» 96	106
» 4. »	» 38,9	39,4		» 100	100
» 5. »	» 37,8	37,6		» 70	84
» 6. »	» 37,0	37,5		» 70	76
» 7. »	» 37,0	37,3		» 40	56
» 8. »	» 37,2	37,6		» 54	68
» 9. »	» 38,0	38,5		» 76	76
» 10. »	» 38,0	38,3		» 90	94
» 11. »	» 38,5	38,8—39,4		» 110	120—130.

Temperaturverlauf ſehr unregelmäßig, bis zum 4. Juni ſteigt die Temperatur von 37,0 bis auf 39,4, dann iſt die Temperatur bis zum 9. normal, an welchem Tage ſie wieder zu ſteigen beginnt, um am Todestage wieder 39,4 zu erreichen. Die Pulsfrequenz iſt etwas regelmäßiger, aber auch nicht ganz der Temperatur coordinirt, am auffälligſten iſt das tiefe Sinken derſelben am 7. (bis auf 40).

41. Fall. Ein 12jähriges Kind wird ins Hoſpital am ſechſten Krankheitstage aufgenommen.

Fiebertabelle.

	Mrgs.	Abds.	M.	A.		Mrgs.	Abds.	M.	A.
19. Juni	T. —	38,0	P. —	56	26. Juni	T. 38,8	38,9	P. 80	88
20. »	» 37,2	37,8	» 56	50	27. »	» 38,8	39,0	» 96	100
21. »	» 37,6	37,9	» 46	56	28. »	» 39,3	39,0	» 90	96
22. »	» 37,7	37,6	» 52	52	29. »	» 38,0	39,3	» 70	80
23. »	» 37,5	38,7	» 52	56	30. »	» 38,6	39,1	» 80	80
24. »	» 38,0	38,5	» 60	60	1. Juli	» 39,1	39,3	» 80	120
25. »	» 38,8	38,9	» 60	80	2. »	» 40,4		P. 180.	

Temperatur ſchwankend, am Morgen des 29. und 30. iſt ſie weit niedriger als am vorhergehenden Tage, ſo iſt dieſelbe auch am 19. Abends höher als an den drei folgenden Abenden. Die Exacerbationen fallen indeſſen immer (mit Ausnahme des 28.) auf den Abend. Am Todestage erhebt ſich die Temperatur auf 40,0 auch nimmt die Pulsfrequenz bedeutend zu, ſonſt iſt dieſelbe in keiner Weiſe der Temperatur coordinirt.

42. Fall. Ein 3jähriges Kind, eingetreten ins Hoſpital am 23. Juni (am Ende der 1. Periode).

Fiebertabelle.

	Mrgs.	Abds.	Mrgs.	Abds.
Den 23. Juni	T. —	39,5	P. —	118
» 24. »	» 39,0	39,0	» 116	100

[41]) Archambault, op. cit. pag. 598. [42]) Ebendaſelbſt. pag. 599.

		Mrgs.	Abds.		Mrgs.	Abds.
Den 25.	Juni	T. 38,8	39,3	P.	80	100
» 26.	»	» 38,7	39,8	»	108	128
» 27.	»	» 39,4	39,0	»	120	96
» 28.	»	» 39,2	39,4	»	126	100
» 29.	»	» 38,7	38,8	»	84	88
» 30.	»	» 38,8	39,3	»	124	138
» 1.	Juli	» 39,1	39,8	»	128	146.

Der Temperaturverlauf ist in diesem Falle ganz unregelmäßig, vom Tageseintritt bis zum Todestage schwankt die Temperatur zwischen 38,7 und 39,8, am Abend des 1. Tages ist sie höher als in den folgenden 2 Abenden, so ist auch die Morgentemperatur des 24. höher als die Temperatur des 25., 26., 29. und 30. Am 24., 27. und 28. constant hohe Temperatur, so auch am Todestage; ihre höchste Ziffer erreicht sie am Abend des 26. und am Abend des Todestages (39,8). Am 24. Morgen- und Abendtemperatur gleich, am 27. Morgentemperatur höher als am Abend, sonst abendliche Exacerbationen. Die Pulsfrequenz ist meist ohne Coordination an die Temperatur, so z. B. zeigt dieselbe am 28. bei einer Temperatur von 39,2 126 Schläge, während sie am Abend desselben Tages bei 39,4 nur 100 Schläge aufweist.

43. Fall. Ein 8jähriges Kind, erkrankt seit dem 23. Mai, ins Spital eingetreten am 31. Mai (2. Periode).

Fiebertabelle.		Mrgs.	Abds.		Mrgs.	Abds.
Den 31.	Mai	T. —	38,8	P.	—	80
» 1.	Juni	» 38,5	39,0	»	72	84
» 2.	»	» 38,4	39,5	»	72	88
» 3.	»	» 38,7	39,8	»	88	96
» 4.	»	» 39,1	39,8	»	84	116
» 5.	»	» 39,2	39,3	»	104	112
» 6.	»	» 39,0	39,2	»	116	120
» 7.	»	» 39,3	39,7—40,8	»	132	148.

Die Temperatur schwankt während dieser Zeit zwischen 38,5 und 39,8, verläuft unregelmäßig und erhebt sich in den letzten 24 Stunden auf 40,8. Vom 4. ab sind sowohl die Morgen- als auch Abendtemperaturen nicht unter 39; mit Ausnahme des 6. fallen die Exacerbationen auf den Abend. Die Pulsfrequenz ist der Temperatur nicht coordinirt.

III. Kategorie.

44. Fall. Ein Mädchen, $9^1/_2$ Jahre alt, hat vor 6 Wochen Masern durchgemacht; vor 8 Tagen schon klagte das Kind über

[43]) Archambault, op. cit. pag. 600. [44]) Handbuch der Kinderkrankheiten. Bd. 5. II. pag. 443.

Kopfſchmerzen, mit denſelben ſtellte ſich auch Erbrechen ein. Am 8. Krankheitstage wird das Kind ins Spital gebracht.

Fiebertabelle.	Mrgs.	Abds.		Mrgs.	Abds.
Den 8. Tag T.	36,5	38,0	P.	64	62
» 9. » »	37,0	37,0	»	66	68
» 10. » »	38,5	37,0	»	78	60
» 11. » »	38,0	40,2	»	74	140
» 12. » »	36,0	—	»	nicht zählbar.	Exitus lethalis.

Section 30 Stunden nach dem Tode.

Sulziges Exſudat zwiſchen Dura und Pia. Venen der letzteren ſehr gefüllt. An der Baſis vom Chiasma NN. opt. bis über die Medulla oblongata hinab eitrige Entzündung der Pia mit reichlichen miliaren Tuberkeln. Ventrikel, namentlich die ſeitlichen, ſtark ausgedehnt durch ſerös-eitriges Exſudat.

Temperaturverlauf äußerſt unregelmäßig, am 9. Krankheitstage iſt die Temperatur niedriger als am vorhergehenden, am 10. Tage iſt ſie am Abend um 1,5 niedriger als am Morgen. Am vorletzten Tage Abends plötzlich auf 40,2 ſteigend, ſinkt dieſelbe am Todestage eben ſo plötzlich auf 36,0. Die Pulsfrequenz iſt nicht ganz der Temperatur coordinirt.

45. Fall. Ein Mädchen, 1 Jahr 4 Monate alt, wird wegen Huſten und Erbrechen, was ſchon 8 Tage gewährt haben ſoll, ins Spital aufgenommen.

Fiebertabelle.	Mrgs.	Mttgs.	Abds.		Mrgs.	Mttgs.	Abds.
Am 1. Tag T.	—	—	37,4	P.	—	—	80
» 2. » »	37,0	—	37,5	»	80	—	74
» 3. » »	37,5	38,0	37,0	»	80	64	64
» 4. » »	36,1	36,5	37,4	»	80	88	104
» 5. » »	37,2	37,0	37,4	»	104	100	114
» 6. » »	37,0	37,0	37,6	»	100	112	108
» 7. » »	37,0	37,0	38,5	»	112	100	122
» 8. » »	37,5	36,5	37,8	»	100	104	100
» 9. » »	40,0	38,7	37,8	»	120	132	122
» 10. » »	37,6	37,5	37,9	»	76	118	92
» 11. » »	um 2 Uhr Morgens		35,4,	P.	76		
» 11. » »	» 5 »		33,6	»	60.		

Die poſtmortale Meſſung ergab nach dem um $5^1/_2$ Uhr erfolgten Tode 34,0, eine Stunde nach demſelben 33,0.

Section 30 Stunden ſpäter.

Dura dem Schädeldach feſt adhärent. In den Sinus viel dünnflüſſiges Blut. Venen der Pia ſtark gefüllt. Gyri abgeplattet, Sulci verſtrichen. Beträchtliches eitriges Exſudat in der Pia der unteren

[45]) Handbuch der Kinderkrankheiten. Bd. 5. II. pag. 458.

Fläche des Cerebellum und über dem Pons. An der Gehirnbaſis iſt dieſer Proceß in etwas geringerem Maße entwickelt und das Exſudat von mehr ſeröſer Beſchaffenheit. An allen dieſen Stellen reichliche Entwickelung grauer Tuberkel. In den ausgedehnten Ventrikeln ein ſeröſer, blutig gefärbter Erguß. Sonſt noch ein alter käſiger Herd im Centrum des rechten unteren Lungenlappens. Bronchialdrüſen verkäſt und reichliche Entwickelung von miliaren Tuberkeln in der Milzkapſel.

Temperaturverlauf ohne Spur von Regelmäßigkeit. In den erſten 3 Tagen Normaltemperatur; iſt dieſelbe am 4. Tage ſubnormal, dann wird ſie wieder bis zum Mittag des 8. Krankheitstages normal, wo ſie abermals bis auf 36,5 ſinkt. Am 9. Tage Morgens ſteigt ſie plötzlich auf 40,0, dann wird ſie wieder normal, bis einige Stunden vor dem Tode ein plötzliches Sinken eintritt, welches bis zum Tode anhält. Die Pulsfrequenz iſt ebenſo unregelmäßig, ohne Coordination an die Temperatur und im durchſchnittlichen Verhältniſſe zu ihr hoch.

46. Fall. Caroline Andreſius, 10 Monate alt, am 19. Januar 1869 im Kinderſpital aufgenommen, ſoll nach Angabe der Eltern erſt ſeit einigen Tagen leidend ſein.

Fiebertabelle.			Mrgs.	Mttgs.	Abds.		Mrgs.	Mttgs.	Abds.
Den 19.	Januar	T.	37,5	37,8	38,2	P.	120	114	112
» 20.	»	»	37,5	37,4	38,0	»	88	100	120
» 21.	»	»	37,5	37,3	37,2	»	120	90	96
» 22.	»	»	36,0	37,4	38,7	»	100	92	98
» 23.	»	»	36,2	37,5	37,7	»	120	100	96
» 24.	»	»	36,5	36,5	36,3	»	116	132	136
» 25.	»	»	36,2	36,0	36,0	»	120	118	102
» 26.	»	»	36,0	37,8	37,0	»	92	120	120
» 27.	»	»	36,0	37,2	37,5	»	110	120	100
» 28.	»	»	37,4	37,0	37,2	»	110	106	100
» 29.	»	»	35,5	36,0	37,0	»	130	118	118
» 30.	»	»	37,0	—	37,2	»	138	—	130
» 31.	»	»	36,0	36,0	36,0	»	130	144	—.

Am 1. Februar 1/4 Stunde post mortem im After gemeſſen 39,3, je 15 Minuten ſpäter 38,0—36,5—35,3.

Section am 2. Februar Vormittags 12 Uhr.

Dura mater blaß, ziemlich glänzend, Gyri faſt vollkommen verſtrichen. An der Baſis des Gehirns ein gallertiges Exſudat, das ſich hinten bis zum verlängerten Mark erſtreckt. An den entzündeten Partieen der Pia mater hie und da kleine miliare Tuberkel. Die Ventrikel ſtark erweitert und mit klarem Serum gefüllt. In

[46]) Jahrbuch für Kinderheilkunde. Leipzig 1868. Bd. II. p. 320.

der linken Chorioidea findet ſich ein gelber feſter Tuberkel von der Größe eines Stecknadelkopfs. Im unteren Lappen der linken Lunge ein friſcher Herd käſiger Pneumonie und Gruppen von miliaren Tuberkeln.

Man braucht nur einen Blick auf die Fiebertabelle zu werfen, um ſich von dem ganz und gar ungeſetzmäßigen Verlauf der Temperatur zu überzeugen. Die erſten 3 Tage weiſen normale oder mäßig erhöhte Temperatur auf, am Morgen des 22. ſinkt dieſelbe auf 36,0, während ſie am Abend desſelben Tages ihre höchſte Ziffer (38,7) erreicht. Am 24., 25. und am Todestage weiſt die Fiebertabelle conſtante ſubnormale Temperatur auf, während an den anderen Tagen normale mit ſubnormalen Temperaturen abwechſeln; post mortem ſteigt die Temperatur bedeutend. Ich werde auf dieſen Fall bei Beſprechung der poſtmortalen Temperaturſteigerungen zurückkommen. Ebenſo unregelmäßig und ohne Spur von Coordination an die Temperatur iſt die Pulsfrequenz, die im Gegenſatz zur Temperatur während der ganzen Krankheitsdauer ziemlich hoch iſt.

47. Fall. K. Leopoldine, 3 Jahre alt, aufgenommen am 9. Mai 1879. Soll bis vor 14 Tagen geſund geweſen ſein.

Fiebertabelle.

	Mrgs.	Abds.	
Den 10. Mai T.	—	40,0	P. 72
» 11. » »	36,7	32,0	» 100
» 12. » »	28,9	28,6.	

Um 3 Uhr Morgens trat der Tod ein. Unmittelbar nach dem Tode 29,8.

Sectionsbefund. Dura mater mit dem Schädeldach zuſammenhängend. Die inneren Meningen über der Convexität der Großhirnhemiſphären zart, an der Baſis mit gallertigem Exſudat infiltrirt. Dieſes Exſudat iſt beſonders ſtark an der unteren Fläche des Pons und um die Medulla oblongata, ferner im Trigonum intercrurale und in der linken Sylviſchen Furche. Im Bereiche der vom Exſudate infiltrirten Partieen der inneren Hirnhäute, aber auch an der oberen Fläche des Kleinhirns, ſowie an der medialen Fläche der Großhirnhemiſphäre zahlreiche, miliare Tuberkel. Die Hirnhöhlen erweitert, mit trübem Serum gefüllt, das Ependym zerfließend weich. Sonſt nur noch in den Lungen zerſtreute, einzelne wenige Tuberkel.

Der Temperaturverlauf iſt ein ganz eigenthümlich unregelmäßiger. Nachdem am Abend des 10. die Temperatur 40,0 erreicht, nimmt ſie von da täglich ab und ſinkt einige Stunden vor dem Tode auf die abnorme Tiefe von 28,6. Die Pulsfrequenz iſt ebenſo unregelmäßig, bei 40,0 iſt die Frequenz des Pulſes nur 72, während dieſelbe bei 32,0 100 aufweiſt.

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[47]) Gnändinger, Jahrbuch für Kinderheilkunde. Bd. 15. p. 459.

48. Fall. M. Anna, $2^1/_4$ Jahre alt, wurde am 25. Juni 1879 mit einem pleuritiſchen Exſudate im Spital aufgenommen, das Exſudat wurde allmälig reſorbirt, ſo daß Anfangs Auguſt nichts mehr von demſelben nachzuweiſen war.

Fiebertabelle.

	Mrgs.	Abds.	
Den 7. Auguſt	T. —	38	
» 8. »	» 38,2	38,0	P. 72
» 9. »	» 37,3		
» 11. »	ſchwankt die T. zwiſchen 37,0 u. 38,0.		P. 92.
» 12. »	Abends ſank die T. auf 32,2.		P. 96.
» 13. »	iſt die T. 31,9. Tod 11 Uhr Vormittags.		

Sectionsbefund. Dura mater mit der Lamina vitrea innig verwachſen. Die inneren Meningen an der Hirnbaſis, im Trigonum intercrurale und den Sylviſchen Spalten leicht ſerös, eitrig infiltrirt, von ziemlich vielen mohnkorngroßen, weißlichen Knötchen durchſetzt. Die Ventrikel des Gehirns erweitert, mit trübem Serum gefüllt, das Ependym zerfließend. Sonſt noch in den Lungen zerſtreute hirſekerngroße, käſige Knötchen, Bronchialdrüſen verkäſt und im unterſten Ileum einige ſekundäre tuberkulöſe Ulcera.

Temperatur ſchwankend, vom 12. ab beſtändiges Sinken derſelben bis auf 31,9. Die Pulsfrequenz iſt ohne Coordination an die Temperatur, bei einer Temperatur von 38,0 iſt die Frequenz des Pulſes 72, dann wieder 92, während ſie bei einer Temperatur von 32,2 96 iſt.

49. Fall. Maria Breindl, 1 Jahr alt, aufgenommen am 19. November 1879, ſoll bis vor 10 Tagen geſund geweſen ſein.

Fiebertabelle.

	Mrgs.	Abds.			Mrgs.	Abds.	
19. Nov.	T. —	38,6	P. 140	23. Nov.	T. 37,5	37,5	P. —
20. »	» 38,2	38,5	» —	24. »	» 37,0	37,7	» 160
21. »	» 38,0	38,6	» 168	25. »	» 38,0	37,4	» klein, ſehr ſchnell
22. »	» 38,2	38,2	» 160	26. »	» 37,0	—	» unzählbar
				»	» 9 Uhr	Vormittags	32,9
				»	» 12 »	Mittags	32,0
				»	» 3 »	Nachmittags	31,0
				»	» 6 »	»	30,1
Unmittelbar vor dem Tode:				»	» 8 »	»	29,4.

Keine poſtmortale Temperaturſteigerung.

Sectionsbefund. Große Fontanelle weit offen. Die inneren Meningen an der Hirnbaſis, namentlich in den Sylviſchen Spalten und im Trigonum intercrurale ſerös-gallertig infiltrirt, von zahlreichen hanfkorngroßen, käſigen Knötchen durchſetzt. Hirnventrikel

[48]) Gnändinger, op. cit. pag. 461. [49]) Ebendaſelbſt. pag. 462.

beträchtlich dilatirt und mit trübem Serum gefüllt. Außerdem in vielen anderen Organen ebenfalls pathologiſche Veränderungen.

Temperatur während des ganzen Verlaufs zwiſchen normaler und wenig erhöhter ſchwankend. Am Todestage ſinkt dieſelbe um 9 Uhr Morgens plötzlich von 37,0 auf 32.9 und fällt von da ab ſtündlich bis auf 29,4. Die Pulsfrequenz iſt hoch und ohne Coordination an die Temperatur.

Folgende 7 Fälle, die ich TURIN's[1]) Abhandlung entlehne, zeigen ähnliches Verhalten wie die letzten 6 Fälle, wenn auch die Temperaturabfälle am Todestage nicht ſo bedeutend ſind wie in den 3 Fällen von GNÄNDINGER.

50. Fall. Gottlieb S., 5 Jahre alt, eingetreten den 20. April. Temperatur während des ganzen Verlaufs unregelmäßig, normal oder ſubnormal, zwiſchen 36,8 und 37,8.

Den 4. Mai Abends 37,6. Den 5. Mai Abends 37,6.
Den 6. Mai Morgens 35,6. Tod 2½ St. nach letzter Meſſung.

51. Fall. Eliſe S., 14 Jahre alt, eingetreten den 28. März, ſeit 8 Tagen Zeichen von Meningitis. Temperatur zuerſt ſubfebril, in den letzten Tagen Abfall.

Den 29. März T. 38,3—38,0. Den 30. März 37,3—38,0.
Den 31. März Morgens 36,5.
Einige Stunden ſpäter trat der Tod ein.

52. Fall. Anna B., 6 Jahre alt, eingetreten den 10. Februar, ſeit 2 Wochen krank. Aufenthalt im Spital mit ſtetigem Sinken der Temperatur.

Den 10. Febr. T. — 38,3. Den 12. Febr. T. 36,9—35,9.
» 11. » » 37—37,5. » 13. » » 33,3—32,4.

53. Fall. Marie B., 3½ Jahre alt, eingetreten den 29. März, iſt ſeit 8 Tagen krank. Im Anfang unregelmäßig febrile, in den letzten Tagen normale und ſubnormale Temperaturen.

Den 7. April T. 35,6—36,2. Den 8. April 36,3—36,5.
» 9. » » 35.2—34,0. Tod in der Nacht.

54. Fall. Thereſe J., 8½ Jahre alt, eingetreten den 10. Februar mit einer Coxitis sinistra; den 15. Januar Reſection; ſeither hohes Fieber. Vom 7. Februar an Symptome einer Meningitis, leichtes Sinken der Temperatur.

Den 17. Febr. T. 38,5—39,0. Den 18. Febr. T. 38,0—36,8.
Den 19. Febr. 8 Uhr Morgens 36,8
» 19. » 1 » Mittags 33,5
» 19. » 5 » Abends 35,4.
Geſtorben 5 Uhr Abends.

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[1]) Op. cit. pag. 23—25.

55. Fall. Emilie B., 6 Jahre alt, eingetreten den 30. Jan.; ſeit 2—3 Wochen krank. Temperatur unregelmäßig, ſchwankt zwiſchen 37,0 und 38,5.

Den 10. Februar T. 38,0—38,2
» 11. » » 37,9—38,8
» 12. » » 37,2—37,8
» 13. » » 36,0.

Tod 2 Stunden nach der letzten Meſſung.

56. Fall. Louiſe R., 4 Jahre alt, eingetreten den 17. November; ſeit 14 Tagen krank. Spitalaufenthalt 2 Tage.

Den 17. November T. 36,5—36,8
» 18. » » 36,3—35,9
» 19. » 2 Uhr Mrgs. 36,7
» 19. » 5 » » 36,3
» 19. » 8 » » 33,8.

Geſtorben um 10 Uhr Morgens.

Dieſe ſtatiſtiſche Zuſammenſtellung zeigt zur Genüge, wie unbeſtändig und unregelmäßig der Temperaturgang bei Meningitis tuberculosa iſt; auch nicht ein einziger meiner Fälle zeigt irgendwelche Geſetzmäßigkeit im Verlaufe der Temperatur, und nimmt es uns nicht mehr Wunder, warum bis jetzt noch immer keine typiſche Curve der Temperatur aufgeſtellt wurde. Das Fieber zeigt eben eine ſolche Inconſtanz, daß an Aufſtellen einer ſolchen nicht zu denken iſt. Aber gerade dieſe Inconſtanz des Fiebers iſt in der differentiellen Diagnoſe ſehr gut zu verwerthen, namentlich manchen Fällen gegenüber mit einem ähnlichen Symptomencomplex wie bei Meningitis tuberculosa, ſo auch manchen Typhusfällen gegenüber wird uns die große Unregelmäßigkeit des Temperaturganges zur richtigen Diagnoſe verhelfen. Im Uebrigen dürfte die Diagnoſe der M. tuberculosa ſehr oft unſchwer zu ſtellen ſein, wenn die Exiſtenz genauer anamneſtiſcher Mittheilungen über das Prodromalſtadium und die eigene Beobachtung weniger Tage der ausgebrochenen Krankheit unſerer Beurtheilung zu Hilfe kommt. Die eigenthümliche Aufeinanderfolge der Symptome, die charakteriſtiſche Art ihrer gegenſeitigen Ablöſung, beſonders der ziemlich ſchnelle Umſchlag des Stadiums der Reizung in das der Lähmung pflegen in den meiſten Fällen recht deutlich hervorzutreten und vor diagnoſtiſchen Irrthümern zu bewahren. Andererſeits jedoch hat man daran feſtzuhalten, daß es keineswegs irgend ein für Meningitis tuberculosa ſpecifiſches Merkmal gibt, und daß auch nicht immer der jeweilig vorhandene Symptomencomplex einen für dieſes Leiden pathognoſtiſchen Werth hat. Die Inconſtanz des Fiebers erſtreckt ſich auch auf die Symptome, und das kliniſche Bild zweier Fälle iſt kaum je nur annähernd das gleiche. Deshalb wird man zur ſicheren Diagnoſe nur durch die Geſammtheit der Erſcheinungen gelangen.

Ich habe nun meine Fälle daraufhin unterſucht: 1. Ob nicht die mehr eitrige Infiltration der Pia, 2. ob nicht die bedeutende Flüſſigkeitsanſammlung in den Seitenventrikeln in irgend welcher Beziehung zur Temperatur ſtehe. Denn ad 1 erwähne ich nur, daß nach BILLROTH's[1]) Verſuch die verſchiedenen Momente zu erörtern, durch welche eine Erhöhung der Körpertemperatur und damit Fieber entſtehe, können unter Anderem auch folgende Umſtände von Bedeutung ſein. a) Es bilden ſich im Blute ohne weiteres Zuthun der Nerven Umſetzungen, die zu geſteigerter Verbrennung Anlaß geben, oder es werden Körper ins Blut aufgenommen, welche ſolche Umſetzungen einleiten und unterhalten; b) das intoxicirte Blut erregt die Nervencentren, und von hier aus entſteht Fieber. Ad 2 erwähne ich die PAGENSTECHER'ſchen[2]) Experimente über Gehirndruck, daß alſo die bedeutende Flüſſigkeitsanſammlung in den Seitenventrikeln, die auf's Gehirn einen Druck ausübt und demgemäß ſeine Functionen alteriren muß, irgendwelchen Einfluß auf die Temperatur haben müßte.

Ich bin jedoch bezüglich beider Unterſuchungen zu keinem poſitiven Reſultate gelangt. Von den Fällen, von denen ich den Sectionsbefund habe, zeigen nur 5 Fälle vom Beginn der Krankheit an hohes Fieber bei eitriger Infiltration der pialen Auskleidungen und ſolchem Exſudat, in 7 Fällen fand ich meiſt ſubnormale, normale oder nur wenig erhöhte Temperatur bei gallertiger oder ſeröſer Durchfeuchtung der Pia und ſolchem Exſudat. Endlich fand ich in der größeren Mehrzahl der Fälle eine mäßig erhöhte oder Normaltemperatur bei ſtark eitriger Entzündung der Pia und eben ſolchem Exſudat. In einem Fall (23.) ſind die erſten 2 Krankheitstage durch hohes Fieber ausgezeichnet, dann wechſelt hohes mit mäßigem Fieber und Normaltemperatur ab, bei ſulzig-eitrigem Exſudat in mäßiger Menge.

Bei der Unterſuchung bezüglich der Flüſſigkeitsanſammlung in den Ventrikeln konnte ich leider zu gar keinem Reſultat gelangen. In allen Fällen ſind zwar die Ventrikel durch Flüſſigkeit ausgedehnt, während die Temperatur, wie ich vorhin gezeigt habe, in den verſchiedenen Fällen den bedeutendſten Abweichungen in der Höhe unterworfen iſt, ſo daß dieſes Ergebniß eigentlich ein direct negatives wäre. Man müßte jedoch, um den wirklichen Einfluß dieſes Factors auf die Temperatur feſtzuſtellen, genaue Meſſungen der angeſammelten Flüſſigkeit vornehmen, um dann die Fälle genau zu vergleichen. In meinen Fällen iſt jedoch auf dieſes Moment kein Gewicht gelegt, ſo daß ich aus dieſem Grunde zu keinem Reſultate gelangen konnte.

Was den Einfluß anderer Affectionen auf das Verhalten der Temperatur bei Meningitis tuberculosa betrifft, ſo iſt, wie ja meine

[1]) Archiv für kliniſche Chirurgie. VI. pag. 429. 1864. [2]) Experimente und Studien über Gehirndruck. Heidelberg 1871.

Fälle beweiſen, die tuberkulöſe Meningitis mit der Verallgemeinerung der Tuberkuloſe ſowie mit den verſchiedenſten pathologiſchen Zuſtänden ſo häufig complicirt, daß ich nicht anſtehe zu behaupten, daß bezüglich der Unregelmäßigkeit des Temperaturganges die mit anderen pathologiſchen Veränderungen complicirte Meningitis tuberculosa keine bemerkenswerthe Differenzen zeigt. Anders verhält es ſich in Bezug auf die Höhe der Temperatur, denn man kann wohl annehmen, daß die Höhe des Fiebers erſtens abhängt von der Maſſe des im Organismus abgelagerten tuberkulöſen Stoffes, zweitens von der größeren oder geringeren pathologiſchen Veränderung der Organe. Die meiſten mit anderen Affectionen complicirten Fälle zeigen deshalb während des ganzen Verlaufs durchſchnittlich bedeutenderes Fieber als die abſolut reinen Fälle von Meningitis tuberculosa. Natürlich fehlt es auch hier nicht an Ausnahmen.

Bezüglich der Abweichungen der Temperatur in den letzten 24—28 Stunden theilte ich ja meine Fälle von vornherein ſo ein, daß dieſelben in dieſer Hinſicht einen raſchen Ueberblick gewähren: 32 Fälle weiſen am Todestage höhere oder ſehr hohe Temperatur auf, 11 Fälle zeigen am Todestage nahezu normale, normale oder mäßig erhöhte Temperatur, während in 13 Fällen am Todestage die Temperatur ſubnormal oder ſehr tief iſt.

Was mag nun die Urſache der rapiden und ſehr hohen Temperaturſteigerung in den letzten Stunden ſein? die doch umſoweniger als eine plötzliche bedeutende Steigerung des Fiebers angeſehen werden darf, als dasſelbe während des ganzen Reizungsſtadiums nicht bedeutend iſt, ohne alle Geſetzmäßigkeit verläuft, und es demgemäß nicht denkbar iſt, daß ſich das Fieber, nachdem bereits Symptome von Collaps eingetreten ſind, ohne allmälige Zunahme zu einem ſolch' hohen Grade ſteigern ſollte.

Auch können wir wohl faſt mit Beſtimmtheit den Einfluß der Convulſionen auf die Temperaturerhöhung, die bei Meningitis tuberculosa zwar häufig vorkommen, ausſchließen. Denn Westphal[1]) weiſt nach, daß die beträchtlichen Temperaturerhebungen bei paralytiſchen Geiſteskranken keineswegs in Beziehung zu den Krämpfen und ihrem Grade ſtehen, auch ohne und bei ſehr geringen Krampferſcheinungen vorkommen, wie denn epileptiſche Anfälle an ſich die Temperatur in keiner Weiſe erheblich ſteigern. Ganz dasſelbe beobachtete Henoch[2]) in ſeinen Fällen, die ich in dieſer Arbeit verwerthet habe. Dieſer Autor berichtet ſogar über einen Fall von anhaltenden toniſchen Krämpfen, in welchem die heftigſten Contracturen aller vier Extremitäten und eine bedeutende Starre der Rückenmuskeln faſt bis zum Todestage anhielten, und dennoch ſank während dieſer Zeit die Temperatur des Kindes von 36,5 auf

[1]) Griesinger's Archiv für Pſychiatrie I. [2]) Op. cit.

32,4, ja am Todestage auf 28,9. Auch ich finde in meinen eigenen, der Heidelberger Klinik entſtammenden Fällen ſehr oft nur leichte Zuckungen und Contracturen oder auch keine Spur von Krampferſcheinungen bei ſehr hohen Fiebergraden, während bei Eintritt von Convulſionen die Temperatur oft unverändert bleibt.

Was den Einfluß terminaler Complicationen mit reſpiratoriſchen Erkrankungen betrifft, ſo können wir zwar in manchen Fällen die plötzliche Erhebung der Temperatur von einer kurz vor dem Tode ſich entwickelnden Lungenaffection ableiten, für die größere Mehrzahl der Fälle müſſen wir jedoch nach anderen Gründen ſuchen, da wir in denſelben acute Affectionen des Reſpirationsapparates durchaus vermiſſen.

Solche rapide prämortale Temperaturſteigerungen wurden auch in vielen Fällen hochfebriler infectiöſer Krankheiten, ſowie auch bei Erkrankungen der Nervencentra angetroffen. Namentlich wurde von Wunderlich[1]) zuerſt darauf hingewieſen, daß am Endſtadium tödtlicher Neuroſen und überhaupt bei mannigfachen Störungen der Nervencentra die Temperatur rapid die außerordentlichſten Höhen erreicht, welche Thatſache ſeither von verſchiedenen Beobachtern beſtätigt worden iſt.

Dann publicirte zuerſt Brodie[2]), daß er in mehreren Fällen von Rückenmarksverletzungen eine beträchtliche Steigerung der Temperatur beobachtet habe. Dieſer erſten Publication folgten dann andere kliniſche Beobachtungen bezüglich des Einfluſſes der Verletzungen des Centralnervenſyſtems auf enorme Steigerung der Temperatur: ſo die von Billroth[3]), Weber[4]), Quincke[5]), Fischer[6]). Dieſe kliniſchen Beobachtungen werden durch die experimentellen Befunde über die Beziehung des Nervenſyſtems zur Körperwärme in hohem Grade unterſtützt. So fand Tscheschichin[7]), wenn er bei einem Kaninchen die Medulla oblongata an der Uebergangsgrenze zum Pons durchſchnitt, daß die Temperatur ſofort zu ſteigen anfing. Auch Schreiber[8]) beobachtete conſtant Temperaturſteigerung nach Verletzung eines gewiſſen Punktes an der Grenze zwiſchen verlängertem Mark und Pons. Dasſelbe Reſultat erzielten Naunyn und Quincke[9]) nach Zerquetſchungen des oberen Markes.

Alle dieſe angeführten Thatſachen werden mit der Annahme eines Moderationscentrums in Zuſammenhang gebracht, welches an der Grenze zwiſchen Hirn und Rückenmark ſeinen Sitz hat, und deſſen Lähmung, ſei es in Folge von Trauma oder in Folge pathologiſcher Zuſtände des Gehirns, eine Temperaturſteigerung bewirke.

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[1]) Op. cit. [2]) Medicochirurgical transactions. XX. 1837. [3]) Langenbeck's Archiv. 1862. [4]) Transactions on the clinical society. London 1868. [5]) Berliner kliniſche Wochenſchrift. 1869. Nr. 29. [6]) Centralblatt für mediciniſche Wiſſenſchaft. 1869. [7]) Reichert's Archiv. 1866. p. 152. [8]) Pflüger's Archiv für Phyſiologie. VIII. 1874. p. 576. [9]) Reichert's Archiv. 1869.

Temperaturabfälle ſind in den letzten Lebensſtunden nicht ſo häufig wie Temperaturſteigerungen. Ich habe zwar in dieſer Arbeit 13 Fälle angeführt, bei denen die Temperatur am Lebensende ſehr niedere Ziffern zeigt, indeſſen ſind aber die 7 Turin'ſchen Fälle das Facit von 38 Fällen, und die 3 Fälle von Gnändinger kamen im Laufe eines Jahres in der Widerhofer'ſchen Kinderklinik vor.

Ein derartiges Sinken der Temperatur geſchieht in vielen Fällen conſumtiver Krankheiten, ſowie auch nach ſtarken Hämorrhagien, ſehr reichlichen Darmentleerungen, wie z. B. bei der Cholera, außerdem kommen ſolche Temperaturabfälle bei Geiſteskranken vor. So berichtet Löwenhardt[1]) über 4 Fälle von Maniacaliſchen, bei welchen Temperaturen von ganz abnormer Niedrigkeit beobachtet wurden. Sie zeigten vor dem Tode 25,0, 29,5, 23,7 und 28,0. (Zwei meiner Fälle (Gnändinger) zeigen ebenfalls abnorm niedrige Ziffern wie 28,6 und 29,4.) Ferner finden wir die verſchiedenſten Beobachtungen in der Diſſertationsabhandlung von Glaser[2]) geſammelt, und erwägt derſelbe alle Umſtände, welche ſubnormale Temperaturen bedingen, worunter die verſchiedenſten Störungen des Nervenſyſtems hervorragenden Antheil nehmen. Er erwähnt auch eines Falles von Meningitis tuberculosa, bei welchem in den letzten 7 Stunden die Temperatur auf 35,0 fiel. Auch Fischer[3]) berichtet über 2 Fälle von Verletzung des Halstheils des Rückenmarks, wobei in dem einen Falle die Temperatur auf 34,0, in dem anderen Falle bis auf 30,0 fiel.

Auch dieſe kliniſchen Beobachtungen werden durch eine Reihe phyſiologiſcher Experimente unterſtützt. So fand Schreiber[4]) conſtanten Temperaturabfall, wenn er von dem bei den Temperaturſteigerungen erwähnten Punkt ein wenig nach vorn mit dem Inſtrument abwich. Auch Tcheschichin[5]) beobachtete ein Sinken der allgemeinen Temperatur, wenn er das Rückenmark an den verſchiedenſten Stellen durchſchnitt. Dasſelbe Reſultat erzielte Leviski[6]) nach Durchtrennung der Varolsbrücke vom Gehirn.

Wir werden daher auch hier wieder auf Störungen jener Partieen des Centralnervenſyſtems hingeleitet, denen die Wärmeregulirung obliegt. Da nun in dem letzten Stadium auch in den anderen Gebieten des Nervenſyſtems Depreſſionserſcheinungen beobachtet werden, ſo muß es ſich ebenſo wie bei den Temperaturſteigerungen um eine Lähmung des die Wärme regulirenden Centrums handeln; es iſt alſo in dieſem Falle das excitocaloriſche Centrum gelähmt.

Unter meinen Fällen zeichnen ſich auch manche dadurch aus, daß die Temperatur post mortem noch zu ſteigen fortfährt. Der-

[1]) Allgemeine Zeitſchrift für Pſychiatrie. XXV. 1868. [2]) Ueber Vorkommen und Urſachen abnorm niedriger Temperaturen. Bern 1878. [3]) Op. cit. [4]) Op. cit. [5]) Op. cit. [6]) Virchow's Archiv. 1869. Bd. 47.

Fiebertabelle.

31. Oct. Mrgs.	T. 39,6	2. Nov. Mrgs.	T. 39,4—39,1 P. 84
» » Mttgs. 12 U.	» 40,1	» » Mttgs.	» 39,5
» » Nchm. 4 »	» 40,1	» » Nchm. 4 U.	» 40,5
1. Nov. Mrgs.	» 38,7	» » Abds. 8 »	» 40,0
» » Mttgs. 12 U.	» 39,2	3 » Mrgs.	» 39,1 P. 90—96
» » Nchm. 4 »	» 40,1	» » Mttgs.	» 40,1
» » Abds. 8 »	» 40,3	» » Nchm. 4 »	» 40,3
» » Nchts. 12 »	» 39,8 P. 74	» » Abds. 8 »	» 39,6.

Den 4. Novbr. Mrgs. T. 38,5, Mttgs. 39,3. P. bis 90. Abds. T. 38,8.

Um 10 Uhr Abends ſtirbt Patient ganz ruhig.

Section den 6. November Vormittags 9 Uhr.

Dura mater prall geſpannt. Pia zeigt auf der hintern Convexität ſtarke Injection, mäßige Trübung und ſeröſe Durchfeuchtung. An beiden Seitenventrikeln ſubpiale Eiteranſammlung. An der Hirnbaſis zeigt die Pia längs den Sylviſchen Gruben ſtark eiterige Infiltration, ebenſo zwiſchen den Längsfurchen. Die Pia und Arachnoides am Pons, Chiasma, Hirnſchenkel intenſiv verdickt, eiterig infiltrirt. Die beiden Seitenventrikel enthalten beträchtliche Mengen einer trüben, mit Eiterflocken durchmiſchten Flüſſigkeit.

Anatomiſche Diagnoſe. Eiterige Baſilarmeningitis, catarrhaliſche Pneumonie beider unteren Lungenlappen. Eiterige Bronchitis.

Wir erſehen nun aus dieſem Falle von eiteriger Baſilarmeningitis, daß der Fieberverlauf ein ganz anderer iſt wie der bei der tuberkulöſen Form. Die Temperatur zeigt vom Beginn der Krankheit ſehr hohe Grade und bleibt auch auf dieſer Höhe bis zum Todestage, an welchem dieſelbe in der Agone auf 38,8 ſinkt. Der Gang der Temperatur iſt ein regelmäßiger mit conſtanten morgendlichen Remiſſionen und abendlichen Exacerbationen. Wir wurden auch von Herrn Hofrath v. Dusch ſogleich darauf aufmerkſam gemacht, daß die Höhe der Temperatur vom Beginn der Krankheit nicht der einer tuberkulöſen Meningitis entſpreche, obgleich die ſonſtigen Symptome auf dieſe Form paßten. Auch die Pulsfrequenz weicht von derjenigen der tuberkulöſen Meningitis ab. Während der Puls, wie ich oben gezeigt habe, in den allermeiſten Fällen von Meningitis tuberculosa enorm beſchleunigt iſt und beim Herannahen des Todes immer noch an Frequenz zunimmt, zeigt derſelbe hier kein ſolches Verhalten. Im Gegentheil, in dieſem Falle iſt der Puls vom Anfang ab auffallend retardirt und zeigt auch in der Agone nur 90 Schläge in der Minute.

Die vorliegende Arbeit ſetzt uns in den Stand Folgendes hervorzuheben:

1. Die Meningitis tuberculosa der Kinder iſt immer, wenn auch nur von einem bruchſtückweiſen Fieber begleitet.

2. Die Temperatur hat einen ganz und gar unregelmäßigen Gang, ſo daß von einer typiſchen Fiebercurve nie die Rede sein kann.

3. Die Temperatur iſt im Durchſchnitt höher oder weniger erhöht, je nachdem die Tuberkuloſe andere Organe, namentlich die Lungen, ergriffen hat oder nicht, oder andere febrile pathologiſche Proceſſe ſich hinzugeſellen, während der Gang der Temperatur immer ein unregelmäßiger bleibt.

4. Am häufigſten iſt die Temperatur am Anfang der Krankheit etwas höher, in der Mitte weniger hoch, während auf das Lebensende meiſt die höchſten Temperaturen fallen, ebenſo wird der Puls gegen das Ende faſt immer beſchleunigt. Indeſſen ſind auch normale und ſubnormale Temperaturen am Lebensende keine Seltenheit.

5. Die Pulsfrequenz iſt abſolut ohne Coordination an die Temperatur und ebenſo unregelmäßig wie dieſe.

6. Im Gegenſatze zur Meningitis tuberculosa verläuft die Temperatur bei der eitrigen Baſilarmeningitis viel regelmäßiger, und iſt auch die durchſchnittliche Höhe derſelben vom Anfang ab eine weit beträchtlichere.

Zum Schluſſe erlaube ich mir, Herrn Hofrath Profeſſor v. Dusch für die gütige Ueberlaſſung des Materials und für die freundliche Unterſtützung meinen beſten Dank auszuſprechen.

C. F. Winter'sche Buchdruckerei.

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Thoracopagus Omphalopagus

by

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The monstrosity described below was secured by Henry Moeller M. D. of No. 240 West 38th St. New York, who kindly furnished the following.

History of Monstrosity born Jan. 26. 91.

Father German, 42 years, well built man with Mitral Stenosis and cardiac Hypertrophy. Mother German-American, 40 years, well built, perfectly healthy. Married 16 years. Menstruated first when 14 years old. Has given birth to one boy and four girls, all well and well formed. There have been also a few miscarriages between these five births, none of which were over two mos. advanced. The youngest is a girl four years old; very bright and good looking. Fathers Mother had twins at first birth; they died before the termination of the first year. After that, she gave birth to four boys and two girls, all of which lived.

Mothers Mother had four girls and two boys, all perfect; the boys died during first year. Fathers Father and Mother reached the ages of 68 and 60 years. Mothers Father and Mother are each 67 years and quite will preserved.

Mother felt peculiar and depressed, while carrying monstrosity, while during all the other pregnancies she felt cheerful and well. All former labors have been normal and not more difficult than the average. On several occasions she got up on third and fourth day, without bad consequences.

At last labor „water broke“ twenty four hours before delivery and all ran off without pains. Twelve hours later, pains commenced

and midwife was called, who had attended to her previous confinements. Found head high up and advancing slowly. After 10 hours, presenting head appeared locked in true pelvis, when my services where required. — Found woman exhausted, labia dry, extremities cold, pulse small and frequent; facies bad. Shape of abdomen peculiar, but different from twin pregnancy. No foetal movements for ten hours. Applied forceps and brought head down incompletely. Now suddenly a tumor appeared over region of bladder; it was diagnosticated as a second head, and utterly prevented further advance of labor. It was impossible to push the advance of head back and as diagnosis of monstrosity was made by exclusion and no foetal heart nor movements were detected, head was removed with considerable difficulty, the neck being well up in vagina. Then hand was introduced past stump and legs brought down. Further progress was hindered until the very large abdomen of second foetus was opened and partly emptied. Now the next legs were brought down and considerable traction and twisting completed delivery after about half an hours very exhausting work. There was but one Placenta, which came away easily.

Laceration had taken place through rectum for about $1\frac{1}{2}$ inches. Parts being very oedematous, did not operate until fifth day, which proved a failure due to very purulent vesical catarrh. After many and copious irrigations of that viscus with 4% Boric Acid solution, a second operation proved successful and patient is now attending to her household duties without inconvenience. The Os uteri is perfect and remarkably small. The external parts are satisfactory and there is a full size new perinaeum. Patient is of medium size, weighs about 125 *lb.* Antero-posterior diameter of pelvis is less than average.

Description of specimen.

Twins, otherwise well-formed, and of about the usual size of full-term twins, are united at the antero-lateral portion of the chest and upper abdomen. The upper limit of the union is just below the shoulder; the lower just below the umbilicus. It consits of a fusion of the thoracic and abdominal cavities of the two children, not simply of an isthmus of skin and subcutaneous tissue.

There is but one umbilical cord, joining the anterior surface of the line of junction near its lower limit. It is of about the usual size.

The heads and four extremities are normal. Both children are females. The accompanying outline drawing represents in a general way their appearance (Fig. 1).

Dissection reveals the following peculiarities.

There is a single sternum, rather broader than usual. There

are two distinct vertebral columns. Fusion, with cartilaginous junction, of the ribs along the posterior portion of the line of union, has occurred.

Fig. 1.

Anterior view of monster, showing union of the two bodies and the single umbilical cord. 1/3.

On opening the abdomen one round ligament is found to run upward from the umbilicus to the liver, carrying a large umbilical vein. Passing downward to the bladders of the two children are two urachi, each carrying its usual hypogastric arteries. The abdominal cavities of the twins connect by a large aperture represented by the

antero-posterior diameter of their bodies and by the distance from the umbilicus to the arch of the diaphragm. The greater part of this opening is filled by a large and many lobed liver, which has

Fig. 2.
Intestine.
Shows the common ileum (A) passing downward to the bifurcation (B), at which point the diverticulum (D) is seen. C = Caput Coli of left child, with its vermiform appendix. Reduce $^1/_2$.

apparently resulted from the fusion of the livers of the two bodies, for, althoug but one gall bladder and bile duct are discovered, there are two quite distinct portal fissures, each conveying branches of its respective portal vein into the liver, and there are two hepatic veins, each joining the ascending cava of its respective foetus. The gall bladder is somewhat larger than usual in a new-born child; the only discoverable bile duct joins the duodenum of the right child.

Fusion of the intestines of the twins has also occurred. Each child has a normally formed oesophagus, stomach and duodenum, but in the upper part of the jejunum the two intestines unite forming a single common tube throughout almost the entire length of the jejunum and ileum. This common tube is much larger in calibre than either of its component guts. About 10 inches above the lower end of the ileum it divides, forming two entirely separate intestines each smaller than the common tube and uniting with its respective colon by a perfectly formed ileo-coecal valve. At the points of this bifurcation of the fused ileum there is a diverticulum about an inch in length resembling in every particular those described as Meckel's diverticula.

The accompanying drawing shows a portion of the common intestine, the point of bifurcation with the diverticulum, the separate ilei, and the ileo-coecal junction of the left child (Fig. 2).

The fused jejunum and ileum just described is supported by a double mesentery. Along the vertebral column of each child the peritoneum folds forward as usual to form the mesentery. The two mesenteries thus formed carry their appropriate mesenteric vessels and approach one another at the intestinal attachment becoming completely fused in places, but remaining separate throughout the greater part of the common tube. At the point of bifurcation of the common ileum each mesentery follows its respective gut.

Each child is provided with a perfectly formed coecum, vermiform appendix, colon, rectum and anus; with a compete set of female generative organs, a spleen, two normally formed kidneys, with their respective ureters, bladders and supra-renal capsules.

A large diaphragm stretches across the bodies of the two twins, separating the abdominal and thoracic cavities.

The common thoracic cavity is occupied by four lungs and a large heart.

Two of the lungs are much larger than the others, those which are anterior and external as regards the monster. The lungs are in two sets, each of which connects in the usual way with its respective trachea. Each is enclosed in a pleural sac.

The heart occupies the centre of the thorax and is evidently the result of fusion of the hearts of the two children. It is broad (5,5 cm) and somewhat flattened antero-posteriorly (2 cm antero-post. diam.) A slight groove on the anterior surface appears to mark the line of fusion of the ventricles of the two hearts, and this same impression of fusion is conveyed by a double apex. The unusual breadth of the ventricular mass, and the above-described grooves give to the heart a shape suggestive of a large double strawberry. Above this large

ventricular mass is a thin walled auricular portion, very broad and supporting four auricular appendices, two of which, rather smaller, overlap the ventricle in front, and two others, larger, are behind and at the side.

From the auriculo-ventricular junction, anteriorly and externally, arise two aortae each of which passes upward and curves backward

Fig. 3.
Anterior view of heart.
1. Ventricular mass; 2. Left aorta; 3. Right aorta; 4. Auricular mass; 5. Auricular appendices from the anterior ventricle; 6. Auricular appendix from the posterior ventricle; 7. Right rudimentary pulmonary artery; 8. Pericardium turned back.

Fig. 4.
Transverse section of the ventricular mass; seen from below.
1. Left ventricle; 2. Right ventricle.

and outward to descend along the vertebral column of its respective child, giving off in its course the usual large branches. These aortae are the only vessels taking origin from the ventricular mass.

On opening the ventricular mass by a transverse cut midway between auriculo-ventricular groove and apex, the appearance of three separate ventricles is presented. This is well shown in the accompanying cut (Fig. 4) Dissection, however, shows the central

and left cavities to communicate freely, forming in reality one large irregularly shaped ventricle. This ventricle opens into the left aorta. The right ventricle becomes prolonged above into a distinct conus arteriosus and opens into the right aorta.

On opening the auricular mass, a transverse partition is seen dividing it into two cavities. These, however, intercommunicate freely, as will presently be described. The anterior of these auricles is somewhat thicker-walled and gives support to the two smaller auricular appendices already mentioned as visible in the front view of the heart. This auricle communicates by two well-formed auriculo-ventricular openings with the left ventricle, each of which is provided with bicuspid valves. At the two sides of this auricle large veins open, coming from the lungs.

The posterior auricle is thinner-walled and rather more irregular in shape than the other, being large at the right and supporting there a large auricular appendix. This auricle communicates with the right ventricle by an auriculo-ventricular opening provided with a well-formed tricuspid valve. Opening into it are two large vessels, one at either side. These are venous trunks formed in each case by the union of the superior and inferior vena cava in each child.

The interauricular septum is very incomplete, two openings in it admitting of free communication between the auricles. These are large, oval in shape, and apear to be the analogues of the foramen ovale in the foetal heart. Along one side of each of these is a thin membranous valve, the Eustachian valve, and it is interesting to note that these valves are attached to the adjacent sides of the two openings in such a way as to direct the blood from the venous trunk nearest it into the anterior ventricle. From this arrangement, and from the character of the vessels opening into these auricles and the nature of the auriculo-ventricular valves in each it is evident that the anterior auricle is the analogue of the left auricle in the normal foetal heart; the posterior, the analogue of the right auricle.

These peculiarities of structure are excedingly well shown in the drawing (Fig. 5). The posterior wall of the auricular mass has been cut away, disclosing a view of the interior of the posterior auricle and the auricular septum. The longer probe passes from the posterior auricle through the right foramen ovale into the anterior auricle, on behind the left foramen ovale, through which it is visible and out through the left pulmonary vein. The shorter probe has been introduced into the posterior auricle through the left common venous trunk. The Eustachian valves may be seen attached to the edges of the partition separating the two foramina.

The absence from this description of any mention of pulmonary

arteries must have been noticed. They are, nevertheless, present, though anomalous and rudimentary. Given off from each aorta at about the usual situation of the ductus arteriosus are arteries of con-

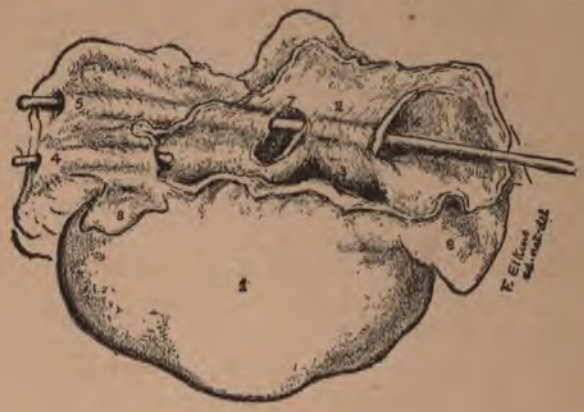

Fig. 5.
Posterior view of heart.
The posterior wall of the auricular mass has been cut away disclosing a view of the posterior auricle and of the interauricular septum.
1. Ventricular mass; 2. Interauricular septum; 3. is placed just above the auriculo-ventricular opening leading to the right ventricle; 4. Common venous trunk of left side; 5. Pulmonary vein of left side; 6. Right foramen ovale with its Eustachian valve along its left border; 7. Left foramen ovale with its similar valve along its right border; 8. Auricular appendices from posteriore auricle.

siderable size, two on the right side, one on the left, which are distributed to the lungs. Soon after leaving the aorta these are joined by a large artery lying just underneath and somewhat behind the arch of the aorta on each side. This vessel passes from this point of union downward, inward and forward towards the auriculo-ventricular junction of the heart, but just before reaching this it ends in a blind extremity, where one of them, the right, is provided with distinct but rudimentary semilunar valves. These vessels are believed to be rudimentary pulmonary arteries whose function has been assumed by the ductus arteriosus and perhaps in part also by enlarged bronchial arteries.

A large and fully closed pericardial sac surrounds the heart and the roots of the large vessels connected with it.

A. Jacobi New York.
May 17th 1891

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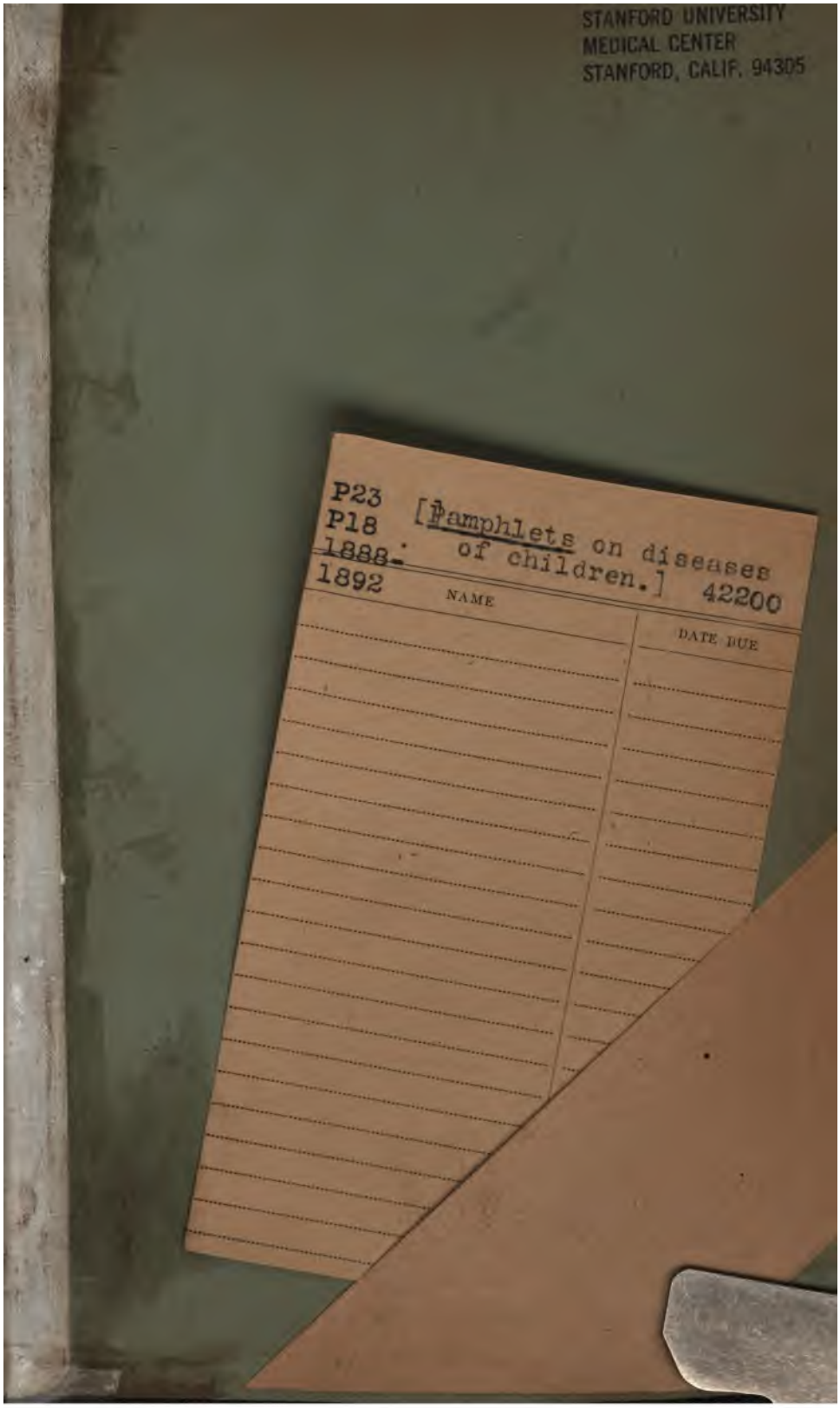
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